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A CLINICAL TRIAL OF ALEVAIRE* IN PULMONARY DISTRESS OF THE NEWBORN INFANT

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DURING the last fifteen years wetting agents have played an important role in industry. Their chief value lies in the fact that they can reduce the surface tension at the intersurface of two liquids of different polarities. Morris[1] has reviewed their chemical structure and pointed out that they consist of:

1. A long chain hydrocarbon radical which is lipophilic or hydrophobic.
2. A short chain SO_4Na radical which is lipophobic and hydrophilic.

The application of these substances at an oil-water interface allows a short chain SO_4Na radical to align with the water while the long chain hydrocarbon aligns itself with the oil or lipoid solution and in this way the surface tension between two such solutions of different polarity is lowered.

Recently these substances have been used in aerosol treatments. Miller[2, 3] pointed out that wetting agents such as "Alevaire" could act as a vehicle for the aerosolization of streptomycin and other drugs. His first series of cases were twelve children with advanced cavitating tubercles of the chest. He emphasized that such a method would enhance the efficiency of streptomycin because the wetting agent would reduce the surface tension at the interface of the caseous cavities. The streptomycin would thus be more soluble in the lipoid caseous material which it is ordinarily unable to penetrate. He noted that Wells[4] had demonstrated that lipoids are present in considerable quantities in caseous tuberculous lesions. All except three of Miller's patients improved, but he pointed out that in so small a series of cases no definite conclusions could be drawn.

None of Miller's cases showed any evidence of toxicity. Gershenfeld and Witlin[5] found no evidence of toxicity when this substance was fed to rabbits. Hall[6] administered Alevaire by aerosol to guinea pigs and found no evidence of toxic damage to their lungs until the aerosol solution concentration exceeded 50 per cent. He then found that the animals suffered from increasing dyspnea, and, microscopically, there was degeneration and desquamation of the alveolar epithelium and fibroblastic proliferation. Miller and Boyer[7] confirmed the lack

*Alevaire (Triton A20, Triton A1339) is a sorbitan polyoxalkylene.

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of toxicity of Alevaire in animals in concentration 100 times greater than that used clinically and they emphasized its inertness, and its stability in acids and alkalines. These workers suggested that Alevaire might be of great value in thinning the thick and viscid secretions present in many of the pulmonary conditions.

Ravenal[8] estimated the effect of Alevaire solution bubbled through saliva, bronchiectatic pus, and amniotic fluid. He found that the viscosities of these solutions as measured by an Ostwald viscometre could be lowered by 10, 19, and 24 per cent, respectively.

ity of 85 to 95 per cent. The plan of study was as follows:

1. All infants demonstrating clinical pulmonary distress sufficient to warrant oxygen or an increase in oxygen flow from the usual 1 to 2 liters were included. Pulmonary distress was assessed by an increase in the rate of respirations, evidence of increasing indrawing of the chest, the appearance of cyanosis which was not previously present, and increasing heart rate and general distress in the child.

2. Alternate case selection was employed. Pairs of twins were considered for the two separate cases.

3. Treatment was begun immediately clinical symptoms of distress became apparent.

TABLE I

GROUP	VERTEX	BREECH	CESAREAN	OTHER
Oxygen	17	7	2	2
Alevaire	12	5	2	3
Total	29	12	4	5

TABLE II

	WEIGHTS IN GRAMS					
	N.W.	<1,000	1,000-<1,500	1,500-<3,000	3,000-<4,500	>4,000
Oxygen	1	3	14	3	4	2
Alevaire	1	1	9	6	3	3
Total	2	4	23	9	7	5

Miller and Boyer[7] suggested that aerosolized Alevaire might be of value in the treatment of neonatal asphyxia. Ravenal[8] was unable to demonstrate its value in cases of experimental hyaline membrane, but claimed remarkable success in cases of neonatal atelectasis, treating eighteen infants, all of whom recovered.

The following clinical trial was carried out between May, 1953, and March, 1954, in the Premature Nursery of the Maternity Pavilion of the Winnipeg General Hospital.

Two groups of cases were studied, one received Alevaire by the Aerosol method of Miller[9] plus oxygen, and the other received oxygen and humid-

4. Alevaire was administered for a twelve-hour period and if there were no signs of improvement it was continued for a further twelve hours.

5. Routine premature care was continued in both groups and all infants were nursed in Armstrong incubators.

6. All cases of gross congenital anomaly were excluded.

FINDINGS

Sixty-two cases were studied. Thirty-four were male, sixteen were female. Twelve infants were excluded from the study, eight because they died within periods of one-half to three hours. Five of these received Alevaire, and three oxygen and humidity. One infant was excluded because he

weighed less than 900 grams and died within three hours. Three infants were excluded because subsequently they were found to have major congenital anomalies.

The modes of delivery of the fifty infants included for the study are listed in Table I.

at birth. Twenty-three cases scored Apgar ratings of less than 7, nine were not rated, and the remaining eighteen had ratings from 7 to 10.

The onset of symptoms was recorded in each group and is shown in Table III. Twenty-nine infants were in respiratory distress from birth,

TABLE III

	HOURS						
GROUP	IMMEDIATE	1	2	3	4	4+	NR
Oxygen	15	0	0	2	3	2	3
Alevaire	14	0	2	0	1	4	4
Total	29	0	2	2	4	6	7

TABLE IV

	OXYGEN SATURATION AND FLOW			
GROUP	2L (30%)	3L (43%)	4L (48%)	4+ (54-60%)
Oxygen	6	8	9	4
Alevaire	5	6	10	2
Total	11	14	19	6

TABLE V

GROUP	LIVED	DIED
Oxygen	16	11
Alevaire	14	9

TABLE VI

GROUP	ATELECTASIS	HYALINE MEMBRANE	PNEUMONIA	PULMONARY HEMORRHAGE	INTRACRANIAL HEMORRHAGE
Alevaire	3	2	3	2	1
Oxygen	3	4	1	1	0

TABLE VII

	1952-1953	1953-1954
Total live births	3,805	3,653
Premature infant births	279	244
Premature infant deaths	50	47
Full-term infant deaths	11	15
Premature death rate per 1,000 live births	12.9	12.9
Total neonatal death rate per 1,000 live births	16.1	16.8

Table II shows the weights of the infants at birth and reflects the fact that the majority of the infants were premature.

Apgar's[10] method of assessment was used to rate the condition of the child

and of those recorded, fourteen occurred at intervals of time from two hours onward.

Of the fifty infants who were considered to fulfill the criteria, twenty-seven received oxygen and high

humidity, while twenty-three were given Alevaire and oxygen. Table IV shows the oxygen flow saturations in both groups as determined by a Beckman analyzer.

The results of treatments in the two groups are shown in Table V. In each of the two groups more than half of the infants died and there is no difference in the mortality rate between these two groups.

One of the objects of this study was to use a clinical basis for pulmonary distress, assuming that over a period of time in this random sample study the number of cases would come to post mortem which would give some indication of how accurate or inaccurate the clinical diagnosis was as regards pulmonary pathology as a cause of death.

Table VI shows a breakdown of the major and pathological causes of death in the twenty infants who died. All were autopsied.

It is interesting to note that in this study only one case was considered to have a major cause of death not related to pulmonary pathology.

Comparison of the same calendar period between 1952 and 1953 with that of the study is shown in Table VII. This table shows that during the study the total neonatal death rate rose slightly while the premature death rate remained constant.

DISCUSSION

In this series, clinical criteria were used to assess pulmonary distress in infants. It will be seen that from the standpoint of delivery, weight, and oxygen flow, both groups are comparable. It is of interest to note that more than half the infants were in immediate respiratory distress at birth. At the beginning of the study an attempt was made to assess the rate of improvement in the infants who survived; this was, however, abandoned as no satisfactory criteria could be established. That the clinical diagnosis of respiratory distress was reasonably accurate is borne out by the fact that all but one of the cases that came to autopsy had some form of pulmonary pathology as a major cause of death.

The findings in this series showed no difference in the survival rate in the group treated with Alevaire. Further, the total neonatal death rate during the study showed no improvement. These findings are in agreement with those of Silverman and Anderson,[11] who studied alternate cases of all infants admitted to the nursery and not specific infants suffering from pulmonary distress. In their series they noted no change in their usual mortality rate as a result of introduction of Alevaire treatment.

Ravenal claimed that Alevaire was responsible for pulmonary expansion in all his infants suffering from primary atelectasis. He did not, however, use any controls to confirm these findings. The present study, however, suggests that Alevaire used with oxygen is no better than oxygen and high humidity in promoting pulmonary expansion.

It seems unlikely that aerosolized wetting agents will in any way benefit infants suffering from the hyaline membrane syndrome. Four infants treated with Alevaire were proved at autopsy to be suffering from this condition, while there were only two with similar autopsy findings in the control group. These find-

ings are in agreement with the experimental work of Ravenal who found no improvement in the lungs of guinea pigs suffering from experimental hyaline membrane after they had been treated with Alevaire.

Although Miller suggested that Alevaire might be of value in neonatal asphyxia, it is hard to understand how these substances could be of value, either in hyaline membrane, and less so in atelectasis, where there is probably very little difference in polarity between the pulmonary secretions and the water-Alevaire mixture which is being aerosolized into the lungs.

In this series, thirty-three out of the fifty infants received oxygen in concentrations of forty-three per cent or greater, which caused considerable anxiety. However, none of these infants developed retrolental fibroplasia, but it is possible that prolonged administration of such an aerosol, using oxygen as the main vehicle, might lead to this complication in small infants. No other toxic effects were noted and this is in agreement with other workers.[3, 6, 8]

Some criticism may be leveled as to why a third group was not given aerosolized water. However, the object of this study was merely to determine whether, other things being equal, the administration of Alevaire as such would be of any value.

CONCLUSION

It is unfortunate that on very inadequate grounds Alevaire has been so widely publicized as an effective therapeutic agent in treating pulmonary distress in the newborn infant. Although this is a small series of cases, it appears that the use of Alevaire in no way improves the outlook of infants suffering from atelectasis and hyaline membrane syndrome in the newborn period.

SUMMARY

A controlled clinical trial was undertaken in infants suffering from pulmonary distress. Sixty-two infants were studied; fifty were considered to be acceptable for analysis. No difference was noted in the survival rates of the infants receiving Alevaire and oxygen. No improvement was noted in the survival rates of infants receiving Alevaire and oxygen compared with those given routine treatment of oxygen and a high humidity. The role of Alevaire in the treatment of pulmonary distress of the newborn infant is discussed.

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PSEUDOMONAS INFECTIONS IN INFANTS ASSOCIATED WITH HIGH-HUMIDITY ENVIRONMENTS

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WITHIN the past few years there has been an increased use of high-humidity environments in the care of newborn infants. Much of this interest in humidity as a therapeutic adjuvant has been occasioned by studies in hyaline membrane disease and other pulmonary disorders of infants.[1-3] Since the initiation of this type of therapy in this hospital, we have found that the use of mists and high-humidity atmospheres may be associated with important and sometimes serious deleterious consequences. It is the purpose of this paper to present one of the complications encountered and to discuss its management.

In the premature and full-term nurseries there were situations under which it was thought advisable to employ mist atmospheres. These included (1) infants delivered by cesarean sections and of diabetic mothers, (2) infants who evidenced signs suggesting hyaline membrane disease or pneumonia, and (3) most premature infants weighing less than 1,800 grams. In all three groups this measure was used as either prophylaxis, or an ameliorative of hyaline membrane disease.

Coincident with the increased use of mist atmospheres for infants was the occurrence of infection due to *Pseudomonas aeruginosa*, whereas previously such infections were a rarity in the nursery. During the preceding two years and throughout the current study there were no changes in the nursery policy regarding the use of antibiotics. In general, a combination of penicillin and streptomycin has been used prophylactically only in those premature infants where there was: (1) maternal ruptured membranes for more than eighteen hours, (2) unsterile delivery, (3) delivery by cesarean section, (4) prolonged resuscitation efforts, (5) suspected pulmonary pathology. Usually these antibiotics were administered for the first seventy-two hours and then discontinued. In the full-term infant, prophylactic antibiotics were not employed unless membranes had ruptured more than twenty-four hours prior to delivery. There have likewise been no changes in the indication for and the techniques of obtaining and examining bacteriological cultures during recent years.

Following the occurrence of a Pseudomonas infection, efforts were made to discover any possible contamination of the incubators or the spray equipment. Cultures of the incubator walls and the water in the reservoir jars showed no growth of Pseudomonas. Sterile distilled water is routinely used in this type of equipment in our nurseries and an investigation disclosed no contamination of the supply bottles. After the first recognized Pseudomonas infection, the incubator in which the patient had been was washed with a 5 per cent solution of acetic acid in addition to the usual cleaning procedures.

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This was established as a routine procedure for all incubators in which mist environments had been produced, despite failures to recover Pseudomonas on cultures from the equipment.

During an approximately one-year period there were thirteen infants in the premature nursery with varying types and degrees of infection in which there was bacteriological evidence for the presence of *Ps. aeruginosa.* The three major portals of entry were the skin, the umbilicus, and the conjunctiva. In two instances the organism was cultured simultaneously from two portals. Positive blood cultures for Pseudomonas were obtained in three infants.

BACTEREMIA

There were three infants with bacteremia, their birth weights ranging from 1,000 to 1,400 grams. In each instance the infant had been placed in a mist atmosphere for three days. Two received penicillin and streptomycin as prophylaxis following maternal ruptured membranes, while the third was on no antibiotics immediately following delivery. Within one week there was evidence of inflammation of the umbilicus, skin, or conjunctiva, and cultures of these sites, as well as of the blood (ante mortem or post mortem) revealed *Ps. aeruginosa.* The following case history is illustrative of these severely infected infants.

CASE 1.—Baby C. was born June 6, 1953, weighing 1,270 grams. He was placed in an Isolette with mist and given 25,000 U. penicillin and 25 mg. streptomycin every twelve hours because of an unsterile delivery. Antibiotics and mist were discontinued after seventy-two hours. At the age of 7 days, the baby was noted to have a purulent yellow discharge in the umbilical stump with surrounding erythema. He was given therapeutic doses of penicillin and streptomycin but died five days later. On the day of death, inflammatory lesions were noted on the scrotum. Cultures of the blood, scrotal lesions, and umbilicus revealed the presence of *Ps. aeruginosa.*

DERMATITIS

Four infants were observed with primary skin lesions. Their birth weights ranged from 1,020 to 1,550 grams. Each infant had been in mist for three days. In two patients a dermatitis was noted three or four days after birth, but in the other two there was no apparent sign of inflammation appearing until twelve to fourteen days after birth. Of the four, only one had been placed on prophylactic antibiotics following delivery. Inflammatory reactions were noted on scattered parts of the body, beginning as erythematous lesions which later became pustular. Later they were ulcerous with accompanying necrosis. *Ps. aeruginosa* was cultured in each case. Similar skin

Fig. 1.—Baby M. with noma of face.

lesions due to *Ps. aeruginosa* have been described in premature infants by Geppert and associates.[4]

CASE 2.—Baby M. was born May 28, 1953, weighing 1,560 grams. He was placed in an Isolette with mist, and on the third day the lips were noted to be swollen and indurated. Culture of the skin taken at this time revealed *Ps. aeruginosa*. He was given 100 mg. Terramycin daily and 2 mg. polymyxin every twelve hours. Mist was discontinued after seventy-two hours, but lesions of the lips progressed into a noma involving the cheeks and nose (Fig. 1). Subsequently, ulcers developed on the trunk and the infant died at age 11 days. Cultures of the blood and cerebrospinal fluid taken post mortem were sterile. An autopsy revealed, in addition to the previously described skin lesions, scattered abscesses on the pleural surfaces of the lungs.

CONJUNCTIVITIS

Three infants, with birth weights ranging from 960 to 1,800 grams, had conjunctival lesions. These infants presented a purulent exudate with marked conjunctival injection occurring one to two weeks after birth. The smallest of this group, described in the following case history, received antibiotics prior to developing conjunctivitis.

CASE 3.—Baby B. was born April 25, 1954, weighing 960 grams. He was placed in an Isolette with mist atmosphere and given 25,000 U. penicillin and 25 mg. streptomycin twice daily for three days as prophylaxis for maternal ruptured membranes. The mist was discontinued after seventy-two hours, and at age 6 days a purulent exudate was noted on the conjunctivae. Terramycin, 50 mg. daily, was given parenterally after cultures were obtained. The cultures of the conjunctivae revealed *Ps. aeruginosa* and the same organism was found in post-mortem blood cultures obtained when the infant died at the age of 9 days.

OMPHALITIS

There were seven infants with umbilical lesions. Their birth weights ranged from 1,020 to 1,750 grams. The umbilicus was the most common site of infection, clinical evidence of infection usually occurring after the infant had been in mist two or three days. The earliest noted sign was erythema about the base of the umbilical stump. Subsequently, a foul, fruity odor appeared along with a greenish-blue discoloration of the stump. The exudate about the umbilicus varied from being thin and scant to thickly purulent. Five of these infants were on no antibiotics following delivery.

CASE 4.—Baby P. was born Nov. 1, 1953, weighing 1,350 grams. She was placed in an Isolette with mist upon arrival in the nursery. After forty-eight hours the cord was noted to be foul-smelling and with a scant exudate. Culture of the umbilical stump revealed *Ps. aeruginosa*. The patient was given 1 mg. of polymyxin intramuscularly every eight hours for three days, and the mist was discontinued. The umbilical lesion healed thereafter, and the patient was discharged from the nursery at the age of two months.

THERAPY

The management of the infants in this study was based on the use of polymyxin B sulfate as soon as infection with *Ps. aeruginosa* was suspected. When an infant showed any clinical evidence of infection, appropriate cultures were taken and therapy with both polymyxin (2 to 3 mg. per kilogram per day in three divided doses intramuscularly) and one of the tetracycline antibiotics (50 mg. per kilogram per day) was instituted. When culture reports confirmed the presence of *Ps. aeruginosa*, polymyxin was continued alone, usually for a three-day period.

If no improvement was noted, the dosage was increased to 4 to 5 mg. per kilogram per day. Urinalyses were performed during the course of the therapy. Polymyxin was also used in the treatment of conjunctivitis in the form of ophthalmic drops (1 mg. per cubic centimeter). Table I summarizes the results in the patients studied. Five of these premature infants (all weighing less than 1,800 grams) died; in each instance the death was considered to have resulted from the infection.

TABLE I. SUMMARY OF INFECTIONS FROM PSEUDOMONAS AERUGINOSA

PATIENT	BIRTH WEIGHT (GRAMS)	MONTH BORN	PORTAL OF ENTRY	BACTEREMIA	INITIAL ANTIBIOTICS*	RESULT
1	1,270	June	Skin, umbilicus	Yes	Yes	Died
2	1,110	April	Skin, umbilicus	Yes	No	Died
3	960	April	Conjunctiva	Yes	Yes	Died
4	1,750	June	Umbilicus	No	No	Died
5	1,560	May	Skin	No	No	Died
6	1,870	May	Umbilicus	No	Yes	Living
7	1,820	July	Conjunctiva	No	No	Living
8	1,550	Dec.	Umbilicus	No	No	Living
9	1,140	June	Skin	No	No	Living
10	1,450	Nov.	Umbilicus	No	No	Living
11	1,020	May	Umbilicus	No	No	Living
12	1,560	Sept.	Umbilicus	No blood culture	No	Living
13	—	Aug.	Conjunctiva	No blood culture	No	Living

*Refers to use of penicillin and streptomycin prophylactically following delivery.

COMMENT

The above cases of infants infected with *Ps. aeruginosa* after being placed in high-humidity environments have been cited to emphasize the importance of the organism in sepsis of newborn infants and its prevalence in nurseries where mist atmospheres are utilized. The ubiquity of *Ps. aeruginosa* and the enhancement of its growth by the increased humidity in its habitat[5] certainly contribute to its role as a pathogenic agent in hospital nurseries.

Though a number of authors[6-11] have shown that conversion of bacteriological flora follows the use of antibiotics, it is not believed that the increase in their application in obstetrical and nursery patients is of primary significance in our observation of an increase in Pseudomonas infection. Only three of the thirteen patients described had antibiotics antecedent to the clinical infection. Moreover, there have been no recent changes in the prescribing of antibiotics on either the obstetrical or pediatric services, and there have been no changes in techniques for obtaining and examining bacteriological cultures. The increased emphasis upon the use of saturated moisture atmospheres has been the one principal change in nursery procedures.

Our management of the problem of Pseudomonas infections depended on (1) early recognition of infection, (2) prompt initiation of polymyxin therapy and its continuation until bacteriological studies no longer indicated the presence of the suspected organism, and (3) limiting the use of mist atmospheres to forty-eight to seventy-two hours.

SUMMARY

Coincident with increased usage of high-humidity atmospheres in the care

of newborn infants, thirteen infants with *Ps. aeruginosa* infections were noted in the premature nursery. The occurrence of such infections was believed to be primarily related to the increased use of such environments. Emphasis is made on the need for early suspicion and recognition of *Ps. aeruginosa* as the etiological agent of infection in infants who have been subjected to mist atmospheres.

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VARIATIONS IN SECONDARY ANTIBODY RESPONSE TO SPECIFIC ANTIGENS

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FOR many years, it has been known that effective immunization of man or animal is based on the fact that the antibody response of the host to an antigen is profoundly modified by past exposure or inoculation. Where there has been no exposure, the response to the first injection is delayed and less marked than the reaction to subsequent injections. The differences between the primary and secondary responses are well illustrated in early studies on serum reactions and vaccinia, and on the antibody response to bacterial toxins, notably diphtheria toxoid.[1]

The production of antibodies has been found to depend not only on the past history of the host but also on the nature of the antigenic stimulus, the method of administration, and other factors, such as the physiological state of the host.[2] The practical aspects of these facts have been incorporated in immunization procedures,[3] the bases of which are not always correctly understood by the physician or the public health worker. Much of the misunderstanding seems to arise from the failure to appreciate fully the importance of the secondary response and the vast differences in the secondary response to various antigens. Consequently, this aspect will be stressed in the present report.

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METHOD OF STUDY

The data to be described were obtained among patients at Letchworth Village, an institution for the feebleminded in New York State, in the course of studies on the prophylactic value of influenza A virus vaccines and of pneumococcal polysaccharides, and on the role of host factors in immunity.[4, 5] The conditions under which these studies were done have been described in previous reports. Since the patients remained at the institution for many years, it was possible to observe them for prolonged periods and to employ as controls uninoculated individuals living under the same environmental conditions. Bleedings were done before and after inoculation as often as desired, and the samples from the same individuals were usually all tested at one time for comparable results.

Fairly complete serological data were obtained in a long-term study of the antibody response of individuals with mongoloid features. These individuals were found to be highly susceptible to certain infections of the respiratory and intestinal tract.[6] An explanation for their unusual susceptibility was sought in a defective antibody-producing mechanism. Relevant data were obtained bearing on

antibody formation following the inoculation of tetanus toxoid and formalinized typhoid bacilli. Since there had been no past inoculations with tetanus toxoid or antitoxin, it was possible to observe the response to primary and secondary stimulation. In the case of typhoid vaccine, however, three doses had been routinely administered subcutaneously on admission, and reinoculations had been made annually by the intradermal route. Consequently, only the effects of reinoculation could be observed for the typhoid vaccine.

Twenty male mongoloids were selected and compared with twenty male individuals without mongoloid characteristics ("nonmongoloid" or "normal"). Both test groups lived in the same cottage with about sixty other feeble-minded patients. They represented the more cooperative individuals in the cottage, and were matched as closely as possible with respect to age, size, degree of mental defectiveness, and duration of residency at the institution. They varied in age from 13 to 36 years, with an average of 21.5 for the mongoloids and 17.0 for nonmongoloid individuals. Mentally, about 75 per cent in each group were classified as imbeciles.

The antigens used in the study were *Salmonella typhosa* in a concentration of 1,000 million organisms per milliliter, and fluid tetanus toxoid, 1 ml. of which protected eight of ten guinea pigs against 10 MLD. The typhoid vaccine was obtained from the New York State Department of Health and the tetanus toxoid from the Lederle Laboratories in Pearl River.

Two series of subcutaneous inoculations of each antigen were given, the first in four weekly doses and the second, a single dose, fourteen months later. In the first series, the dosage of each antigen was 0.5, 0.25, 0.25, and 0.25 ml.; in the second, it was 0.5 ml.

The period of observation lasted thirty-three months. During this period, seventeen bleedings were taken from each patient. Bleedings were done on all subjects on the same day shortly before lunch. They were taken before each injection and at varying intervals after that.

ANTIBODY RESPONSE TO BACTERIAL TOXOID

The response to the inoculation of tetanus toxoid is summarized in Fig. 1, which shows the average concentration of circulating antitoxin per milliliter of serum in the twenty mongoloid and twenty normal individuals in the study. Since none of the individuals had been previously inoculated with tetanus toxoid or antitoxin, there was no evidence of circulating tetanus antitoxin in any of the subjects at the onset of the study. The absence of humoral antibody continued for two weeks following the first dose of vaccine. A rise in titer was observed at the end of three weeks in the normal group and at the end of four weeks in the mongoloid group. The peak concentration of antitoxin in normal and mongoloid groups following the first series of injections was reached in the fourth and fifth weeks, respectively, and was followed by a slow decline over a period of sixty-two weeks. The aver-

age titer following the primary series of inoculations was from ten to twenty times higher in the normal group than in the mongoloid group.

Within each group, there were marked individual differences in response to primary stimulation. Three of the twenty normal individuals and nine of the twenty mongoloids studied had no detectable circulating antitoxin at any time following the initial series of four injections. In the other individuals, appeared to be basic host differences in response to primary stimulation affecting the amount, rapidity, and duration of antibody production. These quantitative differences were related, in part, to the presence or absence of mongoloid features.

The response to reinoculation with 0.5 ml. of tetanus toxoid subcutaneously about fourteen months after the first series of injections is also shown in Fig. 1. A sharp rise occurred in both groups within a week

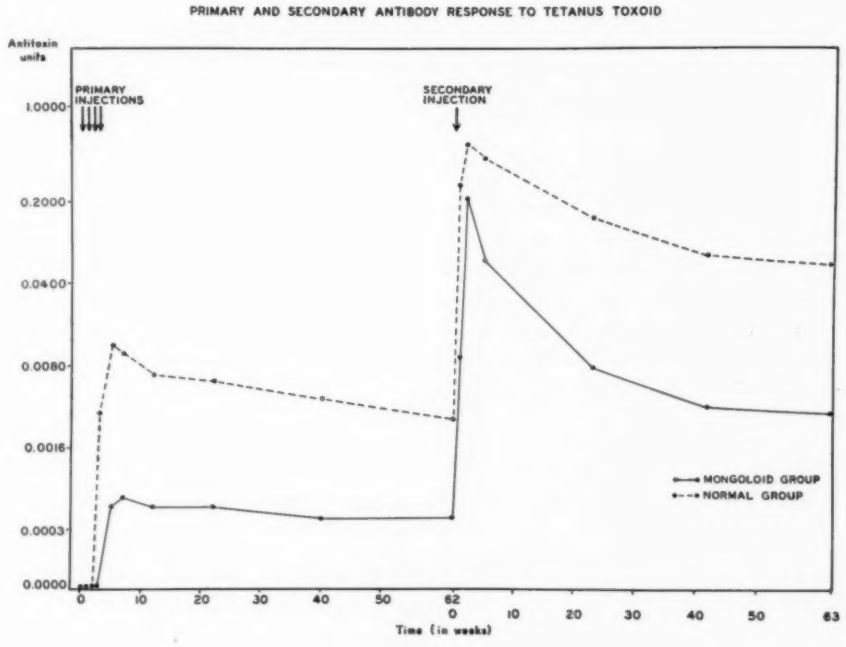

Fig. 1.

humoral antitoxin was detectable in low titer. The maximum for any individual was 0.2 unit per milliliter. It was more commonly observed in the normal group than the mongoloid, and was reached within five weeks by the former and within twelve weeks by the latter. Thus, there after reinoculation, and maximum levels were reached within two weeks. In subsequent weeks, the titers fell sharply at first and then declined slowly. The difference in titer between the normal and mongoloid groups was about threefold at the peak period of antibody production.

After that, there was about a ten- to twentyfold difference between the two groups.

Within each group, there were again marked individual differences in titer. Certain individuals reached concentrations of 1.0 to 5.0 units per milliliter following reinoculation. These high levels were observed more frequently in the normal group than in the mongoloid group, and lasted for a longer period in the former. Most of the individuals, however, had titers of 0.2 unit or less. These lower concentrations were more common in the mongoloid group than in the normal. Only one individual, a mongoloid subject, had no detectable antitoxin following reinoculation. The individual differences within each group noted above are attributable to biological variations which are said to follow a lognormal frequency distribution.[7]

In both groups, the secondary response was much more rapid, more marked, and more long-lasting than the primary (Fig. 1). In other words, those with mongoloid features were capable of an accelerated response which followed the reaction pattern observed in the normal or nonmongoloid group. Furthermore, an accelerated response to reinoculation was observed in all but one individual, regardless of the failure to produce detectable circulating antibody following primary stimulation. It resulted in a fiftyfold increase in circulating antibody over that observed following primary inoculation and a 200-fold increase over the level at the time of reinoculation. Eighteen months later, the average titer was still from five to fifteen times greater than the preinoculation titer. The highly elevated titers long after reinoculation are typical of the secondary response to bacterial toxins.

ANTIBODY RESPONSE TO TYPHOID VACCINE

The data on antibody formation following the inoculation of typhoid vaccine were limited to the effects of reinoculation. The response to four weekly doses at the onset of the study was rapid, occurring within a week and reaching a maximum within three weeks after the first dose or at the time of the fourth dose (Fig. 2). The average increase over the preinoculation titer was four- to sixfold. A similar pattern was reproduced sixty-two weeks later when a single dose of vaccine was given. There were minor differences in the peak level which was reached within two weeks instead of three weeks. But these were attributable to the use of one dose instead of four doses. In other words, the response to the booster dose followed a reproducible pattern characteristic of the antigen regardless of the number of doses.

The response to reinoculation seemed to be similar to that observed for tetanus toxoid in that it was accelerated, but differed from the reaction to toxoid in that it was less marked, the relative increase being four- to sixfold instead of 200-fold. Part of this difference was related to the preinoculation level, for the relative increase following injection varied inversely with the titer before the administration of vaccine. This was quite low for tetanus antitoxin more than a year after primary inoculation.

The average typhoid agglutinin titer in the mongoloid group was lower than that in the group without mongoloid characteristics. The difference between the two groups, taken as a whole, varied from two- to threefold. In both groups the H agglutinin titers were regularly higher than the titers for O agglutinins by about 50 to 100 per cent.[5]

Within each group, there were marked individual differences varying from ten- to twelvefold between lowest and highest limits of titer range. The titers of those with mongoloid features were concentrated about the lower two-thirds of the range of titers, while those without mongoloid characteristics were clustered about the upper two-thirds. However, all individuals, with or without mongoloid characteristics, showed an accelerated response.

CORRELATION BETWEEN RESPONSE TO TETANUS TOXOID AND TYPHOID VACCINE

There appeared to be a low degree of correlation between the extent of an individual's response to a booster dose of tetanus toxoid and his response to typhoid vaccine. This was true for those with and without mongoloid features. The correlated data on circulating antitoxin and O agglutinins within two weeks after reinoculation when the titers were maximal are given in Table I. There was considerable scattering of values at the various titer levels. Although the average antitoxin level seemed to rise somewhat with each increment in O agglutinins, the numbers were small and the reverse did not occur. That is, there was no progressive increase in average agglutinin titer for fixed increments in antitoxin level. The average O agglutinin titer appeared to be the same regardless

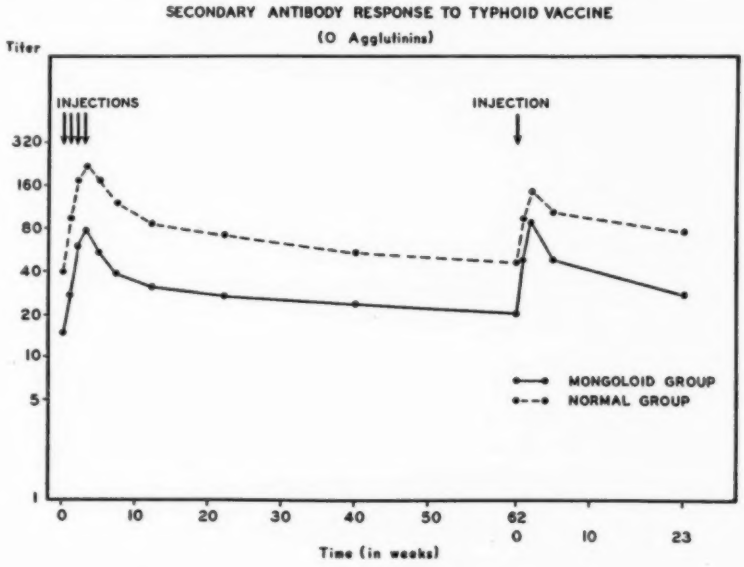

Fig. 2.

of the antitoxin level. Thus, the agglutinin titer of mongoloids possessing the highest levels of circulating antitoxin ranged from 40 to 320, with an average of 125, while the agglutinin titer was 160 for the only mongoloid in the group who failed to respond to any injection of toxoid. Similar unrelated results were obtained among those without mongoloid characteristics. For example, the highest agglutinin titer was 640 in an individual whose maximum antitoxin level was in the lowest quarter of the group. There appeared to be, therefore, no significant association between the maximum concentration of circulating antibodies following the injections of tetanus toxoid and typhoid vaccine. Similarly, when the data on correlated titers sixty-two weeks after reinoculation were examined, there was also no evidence of any association between the amount of antigens, such as diphtheria and typhoid O agglutinins in the same individual.[5]

On the whole, therefore, one could not predict from an individual's response to one type of antigen what his reaction would be to another type of antigen. The lack of correlation between the capacity to produce antibacterial antibodies and antitoxin previously noted was also observed by others in experimental animals.[8] However, a positive correlation was reported in the extent of antibody production to related types of antigens, such as diphtheria and tetanus toxoid, or somatic bacterial and flagellar antigens.[8]

TABLE I. CORRELATION BETWEEN SECONDARY RESPONSE TO TETANUS TOXOID AND TO TYPHOID VACCINE AT HEIGHT OF REACTION

ANTITOXIN (UNITS PER ML.)	O AGGLUTININ TITER						TOTAL	MEAN TITER*
	20	40	80	160	320	640		
Mongoloid Individuals								
0.0000	-	-	-	1	-	-	1	(160)
0.0016	-	1	-	-	-	-	1	(40)
0.008	-	1	1	1	-	-	3	80
0.04	-	-	1	-	-	-	1	(80)
0.2	1	2	3	1	1	-	8	69
1.0	-	1	1	3	1	-	6	125
Total	1	5	6	6	2		20	
Mean titer*	(0.44)	0.12	0.21	0.61	0.98			
Normal Individuals								
0.008	-	1	2	-	-	-	3	65
0.04	-	-	-	-	-	1	1	(640)
0.2	-	1	2	-	4	-	7	160
1.0	-	1	2	1	3	-	7	145
5.0	-	-	1	-	1	-	2	160
Total	-	3	7	1	8	1	20	
Mean titer*	-	0.21	0.44	(2.20)	1.12	(0.09)		

*The results for single individuals only are indicated in parentheses.

PRACTICAL CONSIDERATIONS

The length of time following primary stimulation in which the accelerated response can be elicited could not be determined from the data described. According to various investigations by others, there are indications that it is long-lasting in the case of bacterial toxoid. In one report it was observed as long as ten years after primary injection of

tetanus toxoid.[9] Data on children five to eight years after primary injection revealed a sharply rising titer beginning on the third or fourth day after reinoculation. The sharp rise in titer is within the shortest incubation period commonly observed in tetanus.[10] The rapid secondary response to a single injection many years after primary inoculation seems to preclude the necessity of administering antitoxin following minor injury or of repeating a whole series of inoculations to maintain a high level of immunity long after primary inoculation. However, in extensive trauma or massive contamination where the incubation period is short and case fatality high, antitoxin seems to be indicated despite the excellent results in World War II following the inoculation of toxoid.[11, 12, 13] The use of antitoxin becomes necessary when treatment with tetanus toxoid is delayed too long after injury or when a reliable history of active immunization is not available.

Under ordinary circumstances, immunity can be maintained at a protective level by a small booster dose of tetanus toxoid provided the primary stimulus is adequate and sufficient time has elapsed between primary and secondary injections.[7] In the case of typhoid vaccines, however, where the secondary response appears to be less marked or less prolonged than that described for bacterial toxin, the amount of antigen recommended for reinoculation by the subcutaneous route is larger, and should be repeated more frequently to maintain a high level of immunity. Thus, reinjection has been recommended every year in epidemic or highly endemic areas.[14]

The secondary reaction to typhoid vaccine is reported to be typical of the host response to particulate antigen. Included in this group are not only the bacteria, but also rickettsiae, viruses, and bacteriophages.[15] They vary markedly in their antigenicity. As a group, however, they are said to resemble particulate bacteria rather than bacterial toxins in their antigenic effects. In other words, the striking differences observed between the primary and secondary responses to bacterial toxoid are less distinctive for the particulate vaccines, and more frequent inoculations may be needed to maintain a high degree of protection. Details of dosage and time interval will depend on the nature of the antigen, the risk of natural exposure, and the need for protection under unusual circumstances. These details have to be investigated for each antigen, and have to be tested in the field. For example, a good secondary response was reported following the injection of formalinized poliomyelitis virus.[16] The duration of this effect and its relation to the prevention of paralytic infection under varying natural conditions are still to be determined.

Mongoloid individuals who, as a group, respond poorly to antigenic stimulation can presumably be immunized more effectively in at least two ways. One is to increase their responsiveness by the use of a more potent primary stimulus.[17] The other is to maintain the elevated titer following reinoculation by the use of more frequent booster doses.

RESPONSE TO PNEUMOCOCCAL POLYSACCHARIDES

In recent years, a group of antigens has been identified which induce a primary response but not a secondary response characteristic of other antigens. Reinoculation with these antigens is not followed by a rise in titer until the specific antibody has fallen to a very low level or has disappeared entirely from the blood stream. This type of response was observed following the injection of type-specific pneumococcal polysaccharides.[18] The duration of the negative response to reinoculation of pneumococcal polysaccharides lasted many years, and seemed to depend on the persistence of antigenic stimulation or of antibody formation following primary injection.

Data on the secondary antibody response to the pneumococcal polysaccharides long after primary stimulation were obtained at Letchworth Village in collaboration with Dr. Heidelberger. By means of his quantitative microanalytical method, circulating type-specific antibody nitrogen was found in subjects tested eight years after primary injection of pneumococcal polysaccharides, types I, II, and III in amounts of 0.3, 0.2, and 0.15 mg., respectively (Table II). Reinjection of 50 to 70 μg of the homologous polysaccharides was followed by the reappearance of antibody in those in whom specific antibody had disappeared, and by an increase only in those with small amounts of circulating antibody. All others showed no increase in antibody content following reinoculation. Increases in antibody levels following reinoculation rarely reached the original maximum. In contrast to the small changes in antibodies that usually followed reinoculation, a good response was obtainable in the same individuals injected for the first time with other type-specific polysaccharides, e.g., types V, VII, and VIII.[18]

TABLE II. ANTIBODY RESPONSE TO PNEUMOCOCCAL POLYSACCHARIDES, TYPES I, II, AND III

	MICROGRAMS OF ANTIBODY NITROGEN PER 4 ML. SERUM							
	SECONDARY RESPONSE (EIGHT YEARS AFTER PRIMARY INJECTION)				PRIMARY RESPONSE (CONTROL GROUP)			
WEEKS AFTER INJECTION	CASE NO.	I	II	III	CASE NO.	I	II	III
0	403	0	6	0	402	2	8	1
2		71	7	2		26	17	19
6		49	11	1		34	20	13
0	404	72	51	0	408	0	0	1
2		71	53	6		13	15	18
6		68	57	7		13	15	20
0	405	8	12	17	409	7	10	0
2		14	23	22		34	10	5
6		13	25	23		50	10	6
0	406	16	2	5	410	0	0	0
2		20	13	9		17	42	23
6		19	11	6		10	28	12
0	407	59	27	0	411	0	0	1
2		58	27	9		3	3	6
6		63	29	9		1	1	8

The controls who had never been inoculated with pneumococcal polysaccharides showed a good response to primary injection of all types used, except where there had been evidence of past exposure (Table II). The marked contrast in the response of previously inoculated individuals and of those injected for the first time is illustrated in Fig. 3 for type I pneumococcal polysaccharide.

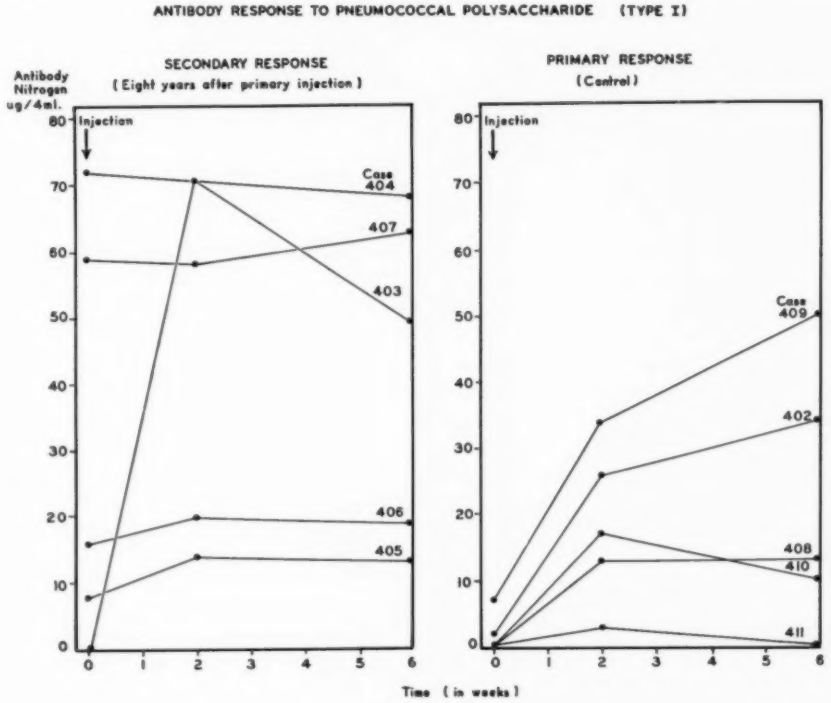

Fig. 3.

The elevated titers in the preinoculation bleeding of some of the controls shown in Table II represent the effect of either natural infection or a cross-reaction due to other causes. The latter may resemble so-called "natural antibodies," which presumably do not prevent an increase in type-specific antibodies following injection (e.g., Case 402, type II, and Case 409, type I).

The explanation for the failure of the host to respond to specific secondary stimulation many years after the primary injection of pneumococcal polysaccharides is not clear. It is said to be related to the persistence of the antigen in the body for a long time, as evidenced by the long duration of circulating antibodies in high concentration following primary injection.[19] At the same time, however, the primary response to new polysaccharides was good. In other words, the lowered or negative secondary response to previously in-

jected pneumococcal polysaccharides was highly specific and, therefore, did not affect the primary formation of antibodies to newly injected polysaccharides.

SUMMARY AND CONCLUSION

Thus, three broad types of antibody response to reinoculation were observed, depending on the antigen used. An accelerated and very marked increase in circulating antibodies commonly associated with a booster dose was seen after the use of tetanus toxoid, and is characteristic of the response to bacterial toxins or toxoids, such as diphtheria and staphylococcus. An accelerated but somewhat less marked secondary reaction was observed following the use of typhoid vaccine, and is said to be typical of antibody formation following particulate antigens like bacteria, rickettsiae, and viruses. Finally, in the case of pneumococcal polysaccharides, no increase in antibodies could be detected following reinoculation until many years after the primary stimulus when the circulating antibody content was very low or nil.

The degree and duration of the secondary reaction also varied with the host. Individual differences were detectable with each antigen used, and are said to follow a lognormal distribution of titers. The individual's secondary reaction pattern to a specific antigen appeared to be reproducible by the use of a single dose only. However, there seemed to be little relationship between his responsiveness to the different types of antigen, that is, to tetanus toxoid and typhoid vaccine.

As a group, the antibody content in the blood stream was lower among individuals with mongoloid features than among those without such characteristics. The relation of these results to hormonal deficiencies reported in the mongoloid is conjectural at present. Electrophoretic analysis of pooled sera revealed comparable levels of gamma globulin in the mongoloid and normal individuals tested.

The foregoing differences in response to reinoculation are not only of scientific interest but also of practical importance in the prevention or modification of clinical infection by means of active immunization. They suggest that different cellular processes might be involved in the formation of various types of antibodies. They serve as a basis for the formulation of an effective immunization program and for its intelligent application under the diversified conditions encountered in practice.

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Some Early Prescriptions

To cure Baldness. Rub the part morning and evening with onions, 'till it is red; and rub it afterwards with honey.

For chin-cough or Hooping-cough. Rub the feet thoroughly with hogs lard, before the fire, at going to bed, and keep the child warm therein,

Or rub the back at lying down with old rum. It seldom fails.

BICKERSTAFF'S BOSTON ALMANACK FOR 1769.

October 6, 1750. I am much afflicted with Toothack.

October 7, had a sorrowful night of Pain.

October 8 . . . I was in much pain; when Mr. Forbush acquainted me with a Remedy used by him with observable success, which he & others communicated the knowledge of; & which tho very Disagreeable yet the more cleanly methods (Blisters &c) used by me, and my affliction great, I yielded freely to—viz Cow dung fryd in Hoggs Fatt—and lay all Night with my Face on it. Despicable as it seems, it gave me Relief. Deo Gratias.

REV. EBENEZER PARKMAN'S DIARY.

To be sold by, Peter Lynch, *near* Mr. Rutger's *Brewhouse, very good Orange Butter, it is excellent for Gentlewomen to comb up their Hair with, it also cures Childrens sore Heads.*

NEW YORK WEEKLY JOURNAL, July 22, 1734.

MEASLES ENCEPHALITIS

I. Prophylactic Effect of Gamma Globulin

Morris Greenberg, M.D., Ottavio Pellitteri, M.D., and Donald T. Eisenstein*
New York, N. Y.

THE value of gamma globulin in preventing or modifying measles has been amply proved since its early demonstration about ten years ago.[1, 2] There is also sufficient experience to indicate that modified measles is a mild disease, not complicated, as a rule, by secondary bacterial infections. There are no data available to indicate whether measles encephalitis is also unusual as a complication of modified measles. Two case reports of encephalitis following measles in children who had been protected by gamma globulin have been published.[3, 4] However, no series of cases sufficiently large for valid statistical evaluation has appeared. This may be due to the fact that encephalitis is an uncommon complication in measles, so that not many cases are seen in one institution.

PLAN OF STUDY

Measles is a reportable disease in New York City. All cases from Jan. 1, 1949, to July 1, 1954, in which encephalitis developed were investigated by an epidemiologist of the Department of Health. In addition to identifying information, the epidemiologist obtained information on the history of the case, symptoms and signs observed in the hospital, the results of spinal fluid examinations, and the condition of the child at discharge. The home was then visited to obtain information, usually from the mother, as to whether the child had received an injection of gamma globulin or other biological during the incubation period of measles. A revisit was made a year or more after the patient's discharge from the hospital, except for the 1954 cases which were revisited one to six months after discharge. A history was obtained from the family or physician about the child's condition, his progress in school, and his relationship with other children. Where a question arose about the child's complete recovery, a physical examination was made by the epidemiologist. No psychologic tests or electro-encephalographic tracings were made.

RESULTS

The incidence of measles encephalitis for the five-year period is given in Table I. There were reported 115,847 cases of measles, of which 165 were complicated by encephalitis an incidence of about 0.14 per cent, or a ratio of 1 case of encephalitis to 702 cases of measles. The ratio was somewhat higher in females (1:611) than in males (1:817), but was about the same for whites and nonwhites. There was an increase in incidence with advance in age, the rate being one and one-half times as great in the 5- to 9-year age group as in the under-5-year group, and three times as high in children 10 to 14 years of age. There were only two cases in infants under one year of age.

From the Bureau of Preventable Diseases, New York City Department of Health.
*Medical Student Fellow of the National Foundation for Infantile Paralysis.

The onset of encephalitis occurred most often about a week after onset of the measles rash, the limits ranging from the day of appearance of the rash to fifteen days later (Table II and Chart I). Onset was usually sudden, with fever, followed by lethargy, convulsions, and stupor which frequently lapsed into coma. Other symptoms were headache, delirium, irritability, mental confusion and disorientation, tremors, and nystagmus. More than a third of the cases developed paralyses of some muscle groups. The fever was high, ranging between 100 and 105° F. in more than 85 per cent of the cases, above 105° F. in about 10 per cent, and below 100° F. in about 4 per cent.

Cervical rigidity was present in more than three-fourths of the cases. The Kernig sign was positive in about 60 per cent and the Brudzinski and Babinski signs in about one-half. The deep reflexes were recorded as absent in 26 per cent and present in 18 per cent, exaggerated in 22 per cent and decreased in about 33 per cent. The superficial abdominal reflexes were not elicited in two-thirds of the patients.

Spinal puncture was performed in 152 cases. None of the fluids yielded any organisms on smear or culture. The cell count was elevated above 10 per cubic millimeter in about 75 per cent of the cases, and lymphocytes predominated in about 90 per cent.

TABLE I. CASES OF MEASLES ENCEPHALITIS BY AGE AND SEX, NEW YORK CITY, JAN. 1, 1949, TO JUNE 30, 1954

AGE	MALES		FEMALES		TOTAL		RATIO
	MEASLES	ENCEPH.	MEASLES	ENCEPH.	MEASLES	ENCEPH.	
0-4	26,529	20	23,865	31	50,394	51	1:988
5-9	28,615	42	26,877	47	55,492	89	1:624
10-14	3,127	8	3,243	11	6,370	19	1:335
15	1,390	3	2,201	3	3,591	6	1:599
Total	59,661	73	56,186	92	115,847	165	1:702
Ratio	1:817		1:611		1:702		

TABLE II AND CHART I.—INCUBATION PERIODS OF MEASLES ENCEPHALITIS FROM DAY OF APPEARANCE OF MEASLES RASH, NEW YORK CITY, JUNE 1, 1949 TO JUNE 30, 1954

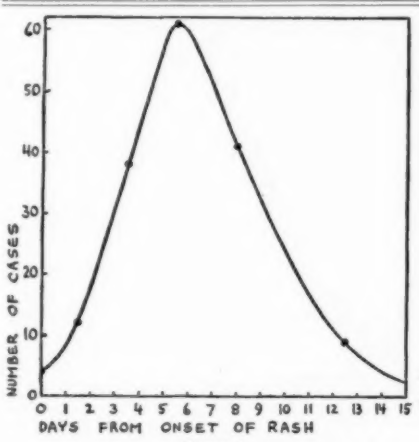

Chart I.

INTERVAL BETWEEN ONSET OF RASH AND OF ENCEPHALITIS IN DAYS	NUMBER OF CASES
Same Day	4
1-2 days	12
3-4 days	38
5-6 days	61
7-9 days	41
10-15 days	9
Total	165

TABLE III. SEQUELAE, DEATHS, AND RECOVERIES IN 165 CASES OF MEASLES ENCEPHALITIS, NEW YORK CITY, JAN. 1, 1949, TO JUNE 30, 1954

	MALE					FEMALE					TOTAL				
Age	0-4	5-9	10-14	15+	Total	0-4	5-9	10-14	15+	Total	0-4	5-9	10-14	15+	Total
Cases	20	42	8	3	73	31	47	11	3	92	51	89	19	6	165
Sequelae:															
Number	0	5	1	0	6	1	7	1	0	9	1	12	2	0	15*
Per cent	0	11.9	12.5	0	8.2	3.2	14.9	9.1	0	9.8	2.0	13.4	10.5	0	9.1
Deaths:															
Number	3	3	0	0	6	10	8	0	0	18	13	11	0	0	24
Per cent	15.0	7.1	0	0	8.2	32.3	17.0	0	0	19.6	25.5	12.4	0	0	14.5
Recoveries:															
Number	17	34	7	3	61	20	32	10	3	65	37	66	17	6	126
Per cent	85.0	81.0	87.5	100.3	83.6	64.5	68.1	90.9	100.0	70.6	72.5	74.2	89.5	100.0	76.4

*Four of these could not be traced. It is assumed that their sequelae persisted after discharge from hospital.

Sugar was normal in all, and protein was elevated above 50 mg. per 100 c.c. of fluid in 60 per cent of the cases.

There were 24 deaths, a fatality of 15 per cent. It was twice as high in girls as in boys, and twice as high in children under 5 as compared with those between 5 and 10 years of age. There were no deaths in the older age groups (Table III).

On discharge from the hospital, 110, or 67 per cent had made a complete recovery. On revisit about a year later, 28 could not be traced. Of the rest, all but one had remained normal. One child was found to be slightly retarded. Whether this retardation existed at the time of discharge but was not recognized, or whether there was a true retardation after discharge, is not clear. Of the 31 discharged with one or more sequelae, 22 had paralysis or paresis of muscles, 6 mental or personality changes, three increased deep tendon reflexes, and two convulsions. When revisited a year later, 16 had made a complete recovery, 4 could not be traced and, 11 still had sequelae. The total number of complete recoveries was 126 or 76 per cent (Table III). It is interesting to note that although mortality was highest in the youngest age group, sequelae were lowest. They were about the same in boys as in girls.

Inquiry was made about the prophylactic use of gamma globulin prior to onset of measles in the cases that developed encephalitis. Only one child had received any. This was a boy, 5 years of age, who was suffering from cerebral palsy. He was exposed to measles in a sibling and was given 2 c.c. of gamma globulin. He developed typical measles about two weeks later, and on the same day that the rash appeared he showed signs of cervical rigidity. A spinal tap yielded clear fluid with 57 lymphocytes to the cubic millimeter and 47 grams protein to 100 c.c. of fluid. He recovered from the encephalitis.

There are no figures available for the number of children that received prophylactic injections of gamma globulin and developed modified measles. However, one can arrive at an estimate by a knowledge of the amount of gamma globulin used in the city for that purpose. From Jan. 1, 1949, to July 1, 1954, there were distributed by the Department of Health to physicians 633,928 c.c. of gamma globulin for use in contacts to cases of measles. Only one dose per contact was allowed and the distribution was limited to children between the ages of 6 months and 5 years, living in the household of a case of measles. An exception was made for children who were contacts to a case on the wards of a hospital. Here it was allowed for children of any age, and those between 5 and 10 years of age received two doses each. In May, 1954, the distribution was liberalized, and susceptible contacts of all ages were permitted to obtain gamma globulin. In addition to the free supply, physicians were able to purchase gamma globulin on the market, without restrictions, until 1953. In 1953 and 1954 all commercial supplies were purchased by the National Foundation for Infantile Paralysis, and contributed to the national pool, so that none remained on sale. Two pharmaceutical firms sold gamma globulin on the market from 1949 to 1952. One of these sold 85,800 c.c. in the city. The other was unable to sup-

ply figures of sales to the city, but since it is a popular concern, and since its sales antedated those of the other company, there is reason to believe that at least as much was sold by this company. The total amount sold in the city for the four years was, therefore, approximated 170,000 c.c.

The public supply went to contacts under 5 years of age, except in hospitals. In the period covered, 115,847 cases of measles were reported, of which 50,394 were in children under 5 years of age. More than 300,000 doses of gamma globulin were distributed to household and hospital contacts of cases. A considerable number of the injected children must have developed modified measles, yet only one case of encephalitis occurred in a child that had been injected. About 86,000 doses were sold in the city in four years. This went for contacts above the age of 5 years. In those four years there were reported 71,705 cases of measles, of which about 38,000 were 5 years of age or older. It appears probable that a considerable number of these or of unreported cases received gamma globulin. Yet not a single case of encephalitis in this age group was reported. While it is probable that many cases of measles went unreported, it is not probable that many cases of encephalitis failed to come to the attention of the Department of Health, particularly since almost all such cases were hospitalized.

COMMENTS

This study was projected to determine whether gamma globulin has any value in preventing the encephalitic complications of measles. Its effect in the prevention and modification of measles has been amply studied, and there is sufficient clinical evidence that modified measles is exempt from the usual bacterial complications. No data have been available, however, to indicate whether encephalitis is less frequently a complication of modified than of unmodified measles.

All reported cases of measles encephalitis from the beginning of 1949 to July 1, 1954, were investigated epidemiologically. A total of 165 cases was recorded, a ratio of one case to 702 cases of measles. It was somewhat higher in females (1:611) than in males (1:817) and was the same in whites and nonwhites. The incidence of the complication increased with the increase in age of the patients with measles; and, as was to be expected, few cases occurred in infants under one year of age. The case fatality was 15 per cent. In contrast to the incidence of the complication, the younger the age, the greater was the mortality. No deaths occurred in children 10 years of age or older.

The incubation period, symptoms, and signs of the encephalitis following measles differed little from encephalitis following other infectious diseases. Of the patients that survived 67 per cent were normal on discharge from the hospital. On recheck a year or more later an additional 9 per cent appeared normal, so that 76 per cent of the patients with encephalitis made complete recoveries, 7 per cent remained with sequelae, 2 per cent were not traced, and 15 per cent died. Previously reported groups of cases show considerable variation in mortality and recovery rates depending, to a considerable extent, on the size of the group and the extent to which the cases were followed. The average

mortality was 15 per cent and the average rate of sequelae was 30 per cent, with 10 per cent not recorded or followed.[5]

Only one of the 165 patients with encephalitis had received a prophylactic dose of gamma globulin prior to the onset of measles. Since more than 300,000 doses of gamma globulin were distributed during the period of study for use in contacts under 5 years of age, there is reason to believe that many cases of modified measles occurred among the contacts to the 50,000 reported cases in that age group and the many unreported cases. Yet only one case of encephalitis occurred in this group, whereas 50 cases occurred among the uninjected ones. About 86,000 doses were sold in the city for the use of older children in the four-year period of 1949 through 1952. Yet not one case of encephalitis occurred among those injected, but 80 cases occurred among the 38,000 reported cases of measles that were not injected. The conclusion appears justified that encephalitis is a rare complication of measles modified by gamma globulin.

CONCLUSIONS

1. Over a period of five and one-half years, 165 cases of encephalitis occurred as a complication of 115,847 reported cases of measles in New York City. The ratio of cases of encephalitis to cases of measles was higher in females than in males, but was about the same in whites and nonwhites. It was one and one-half times as high in children 5 to 9 years of age and three times as high in those 10 to 14 years of age as in those under 5 years of age.

2. The case fatality rate was 15 per cent. It was twice as high in females as in males and twice as high in children under 5 years of age as in those 5 to 9 years of age. There were no deaths in older children.

3. Sequelae occurred in about 9 per cent of the cases, divided equally between boys and girls. They occurred about six times more often in those 5 to 14 years of age than in those under 5 years. There were none in children 15 years of age, or older.

4. All but one of the cases of encephalitis occurred in children who had not received prophylactic doses of gamma globulin prior to onset of measles, although there were distributed during the period of the study about 300,000 doses of gamma globulin for children under 5 years of age; and, during the first four years of the study, about 85,000 doses for those 5 years of age or older.

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MEASLES ENCEPHALITIS

II. Treatment With Gamma Globulin

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ATTEMPTS to treat diseases caused by small viruses, such as poliomyelitis and measles, with immune serums or their fractions have not met with success.[1, 2] The effectiveness of such serums in prophylaxis has been amply demonstrated.[3, 4, 5] It is true that convaleseent serum and gamma globulin have been shown to be of value in the treatment of preeruptive measles. This is the stage when viremia is probably present.[6] No value from the use of the serums was found after the rash appeared.[2, 7, 8]

Theoretically, it is improbable that a specific antiserum will be effective in treatment unless given early in the course of the disease when the causative organism or its toxin is still circulating in the blood. Once the virus has become attached to the cell it cannot be neutralized by antibody. In measles, the median day of onset of the complicating encephalitis occurs, about six days after the beginning of the rash.[9] By then the upper respiratory discharges contain no virus and the antibody titer of the serum is fairly high.[6] It is difficult to see how the use of gamma globulin could affect the course of the complications or determine their outcome.

Results of treatment in single cases are worthless for evaluation. While it is generally true that cases with severe symptoms and signs have a higher mortality rate than those with mild symptoms, the outcome in an individual case is not always dependent on the severity of symptoms at onset. Patients in stupor, coma, or continued convulsions at onset often make complete recoveries.[10, 15] An example of an unusually severe case with recovery is the following:[11]

J. A., 6-year-old white female, was admitted to the hospital on April 3, 1954, with a history of a measles rash three days before admission. On the evening prior to admission, her temperature rose to 103° F.; she became stuporous and lapsed into coma. She continued in a comatose state for three weeks, and remained stuporous for another four weeks. On April 8, her temperature rose to 107° F., and she was cyanotic and dyspneic. An intubation and then a tracheotomy was performed. On April 10, an x-ray film of the chest showed some infiltration in the upper left lung, which was interpreted as aspiration pneumonia. The following day she was placed in a respirator on account of shallow breathing, but she was soon taken out of it because she breathed against the machine. The child received oxygen over the tracheotomy opening. She continued in coma until April 24, when her sensorium began to show signs of improvement, but her tem-

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perature continued elevated until April 30, and she remained stuporous for four weeks. The tracheotomy tube was removed on May 9. Nasal tube feedings were discontinued three days later. The child showed extreme emotional lability, crying out frequently and showing numerous athetoid movements. On May 17, the athetoid movements had disappeared, irritability was still marked, but memory and intellectual function seemed better. Improvement continued and the child was discharged on June 1, 1954. Except for some possible personality changes and enuresis, no permanent sequelae were noted. When seen on June 11, 1954, the only abnormality noted was a left Babinski sign and slight widening of the base in walking.

This child received no gamma globulin. She received cortisone, total 325 mg., for the first four days and ACTH, 25 mg., on the fifth day. Penicillin was used as a covering antibiotic, and the rest of the treatment was symptomatic. Although the child has not been observed long enough to determine how complete her recovery is, there is little doubt that she has made a remarkable recovery without benefit of any specific treatment except for cortisone and ACTH in the first five days.

Plasma or its fractions have been used for the treatment of measles encephalitis in a few small series of cases with no apparent benefit.[10, 12, 13] Recently a report has appeared in which the statement is made that gamma globulin was effective in reducing the mortality rate and the incidence and severity of sequelae from measles encephalitis in a series of forty-one cases, of which twenty-seven patients were treated and fourteen untreated.[14]

In a projected study on the value of gamma globulin in the prevention of measles encephalitis, begun in 1949, epidemiological data were collected, which included methods of therapy. All reported cases in the City of New York were investigated, and patients were revisited a year later (except in 1954 when they were seen again one to six months later) to determine their physical and mental condition.[9] There were 165 cases of encephalitis in the investigation, but this study is limited to the 159 patients under the age of 15 years, since none above that age was treated with gamma globulin or died. Of these, 51 patients were treated with gamma globulin and 108 were not treated with it. The patients with encephalitis receiving gamma globulin included 14 treated by one of us, all but two of whom were in the Willard Parker Hospital. The rest were treated in the other communicable disease hospitals of the city, and we are indebted to their superintendents for permission to inspect the charts.

Table I lists the cases of encephalitis by age and according to treatment with gamma globulin. Treatment was given in the acute stage of the disease. It was begun on the day of onset in 50 per cent of the cases, on the day after onset in 26 per cent, and in the first four days in 94 per cent of the cases treated. It will be noted that the fatality rates of treated and untreated cases were 13.7 and 15.7 per cent, respectively. The sequelae in the two groups were 11.8 and 9.2 per cent, respectively. The differences in rates of both are statistically insignificant. If deaths and sequelae are combined, the rates were 25 per cent for the treated and the untreated.

There might be differences in therapeutic results in different age groups. In Table I the results are arranged according to age. In the youngest age group deaths were 27 per cent in the treated and 25 per cent in the untreated; in the 5-to-9-year age group, the death and the sequelae rates were 20 and 12.5 per cent for the treated and untreated patients, respectively. None of the differences were of statistical significance.

Five children admitted with a diagnosis of measles encephalitis died on or soon after admission, before treatment of any sort could be adminis-

TABLE I. PATIENTS WITH MEASLES ENCEPHALITIS TREATED WITH AND WITHOUT GAMMA GLOBULIN, NEW YORK CITY, JAN. 1, 1949, TO JUNE 30, 1954

	NO GAMMA GLOBULIN				TREATED WITH GAMMA GLOBULIN			
AGE	CASES OF ENCEPHALITIS	SEQUELAE	DEATHS	DEATHS AND SEQUELAE	CASES OF ENCEPHALITIS	SEQUELAE	DEATHS	DEATHS AND SEQUELAE
0-4								
Number	40	2	10	12	11	0	3	3
Per cent	100	5.0	25.0	30.0	100	0	27.3	27.3
5-9								
Number	60	7	7	14	30	4	4	8
Per cent	100	11.6	11.6	23.3	100	13.3	13.3	26.7
10-14								
Number	8	1	0	1	10	2	0	2
Per cent	100	12.5	0	12.5	100	20.0	0	20.0
Total								
Number	108	10	17	27	51	6	7	13
Per cent	100	9.2	15.7	25.0	100	11.8	13.7	25.5

TABLE II. PATIENTS WITH MEASLES ENCEPHALITIS TREATED WITH AND WITHOUT GAMMA GLOBULIN, EXCLUDING PATIENTS WHO DIED ON OR SOON AFTER ADMISSION, NEW YORK CITY, JAN. 1, 1949, TO JUNE 30, 1954

	NO GAMMA GLOBULIN			TREATED WITH GAMMA GLOBULIN		
AGE	CASES OF ENCEPHALITIS	SEQUELAE	DEATHS	CASES OF ENCEPHALITIS	SEQUELAE	DEATHS
0-4						
Number	36	2	6	11	0	3
Per cent	100	5.5	16.7	100	0	27.3
5-9						
Number	59	7	6	30	4	4
Per cent	100	11.9	10.2	100	13.3	13.3
10-14						
Number	8	1	0	10	2	0
Per cent	100	12.5	0	100	20.0	0
Total						
Number	103	10	12	51	6	7
Per cent	100	9.7	11.7	100	11.7	13.7

13.3 and 11.6 per cent for treated and untreated patients, respectively. In the 10-to-14-year age group there were no deaths, and the sequelae rates were

tered. Four were under 5 and one was 6 years of age. If we omit these cases from the comparison of treated and untreated cases, the mortality for

patients not treated with gamma globulin is reduced to 11.7 per cent (Table II).

Cases of encephalitis vary from those that present comparatively mild symptoms to those that have profound neurological disturbances. Comparisons between the treated and untreated should be made separately for the severe and the milder cases. There are no quantitative criteria to separate the two groups, and reliance on clinical impressions is unsatisfactory. We have divided the two groups into mild and severe, and have placed in the latter group the patients who were in stupor or coma or had convulsive episodes during the acute stage. The results are given in Table III. There occurred 7 deaths among 45 severe cases treated with gamma globulin (15.5 per cent) and 15 deaths among 72 severe cases not treated with gamma globulin (20.8 per cent). The number of patients with sequelae in the treated and untreated groups were 6 (13.2 per cent) and 8 (11.1 per cent), respectively. There are no statistically significant differences in the two groups on the basis of deaths, sequelae, or both. If the 5 patients that died on or soon after admission are omitted, the differences between the specifically treated and untreated cases are even smaller (Table IV).

It is possible that the amount of gamma globulin administered might make a difference in the results. In Table V, the severe cases (exclusive of the patients that died on or soon after admission) are divided into three groups: those that received no gamma globulin, those that received up to 20 c.c., and those that received 21 to 160 c.c. There were 67 cases among the untreated, with 10 deaths and 8 with sequelae; 21 among those receiving the smaller amount of gamma globulin, with 5 deaths and 3 with sequelae, and 24 among those receiving the larger amount, with 2 deaths and 3 with sequelae. The combined deaths and sequelae in the three groups are 26.8, 38.1, and 20.8 per cent, respectively. None of the differences are of statistical significance.

TABLE III. PATIENTS WITH SEVERE MEASLES ENCEPHALITIS TREATED WITH AND WITHOUT GAMMA GLOBULIN, NEW YORK CITY, JAN. 1, 1949, TO JUNE 30, 1954

	NO GAMMA GLOBULIN				TREATED WITH GAMMA GLOBULIN			
AGE	CASES OF ENCEPHALITIS	SEQUELAE	DEATHS	DEATHS AND SEQUELAE	CASES OF ENCEPHALITIS	SEQUELAE	DEATHS	DEATHS AND SEQUELAE
0-4								
Number	26	2	8	10	10	0	3	3
Per cent	100	7.7	30.8	38.5	100	0	30.0	30.0
5-9								
Number	43	6	7	13	26	4	4	8
Per cent	100	13.9	16.3	30.2	100	15.4	15.4	30.8
10-14								
Number	3	0	0	0	9	2	0	2
Per cent	100	0	0	0	100	22.2	0	22.2
Total								
Number	72	8	15	23	45	6	7	13
Per cent	100	11.1	20.8	31.9	100	13.3	15.5	28.8

In evaluating the results of therapy it is not enough to point to the remarkable results achieved in one or a few cases with a certain form of therapy. Such results might have been due to chance. It is essential to compare the treated with a similar group of untreated cases and to observe whether significant statistical differences exist in the results achieved by the two. In the present investigation a sufficiently large number of treated and untreated patients of similar ages were compared and the results indicate that whether the comparison is made for the two groups as a whole or by specified age groups, or by amount of gamma globulin administered or by severity of case, no essential difference was observed between the treated and untreated, whether this was based on deaths or on sequelae or on both. The

TABLE IV. PATIENTS WITH SEVERE MEASLES ENCEPHALITIS TREATED WITH AND WITHOUT GAMMA GLOBULIN, EXCLUDING PATIENTS WHO DIED ON OR SOON AFTER ADMISSION, NEW YORK CITY, JAN. 1, 1949, TO JUNE 30, 1954

AGE	NO GAMMA GLOBULIN			TREATED WITH GAMMA GLOBULIN		
	CASES OF ENCEPHALITIS	SEQUELAE	DEATHS	CASES OF ENCEPHALITIS	SEQUELAE	DEATHS
0-4						
Number	22	2	4	10	0	3
Per cent	100.0	9.1	18.2	100.0	0.0	30.0
5-9						
Number	42	6	6	26	4	4
Per cent	100.0	14.3	14.3	100.0	15.4	15.4
10-14						
Number	3	0	0	9	2	0
Per cent	100.0	0	0.0	100.0	22.2	0.0
Total						
Number	67	8	10	45	6	7
Per cent	100.0	11.9	14.9	100.0	13.3	15.5

TABLE V. PATIENTS WITH SEVERE MEASLES ENCEPHALITIS TREATED WITH DIFFERENT DOSES OF GAMMA GLOBULIN, AND WITHOUT GAMMA GLOBULIN, EXCLUDING PATIENTS WHO DIED ON OR SOON AFTER ADMISSION, NEW YORK CITY, JAN. 1, 1949, TO JUNE 30, 1954

AGE	NO GAMMA GLOBULIN			GAMMA GLOBULIN UP TO 20 C.C.			GAMMA GLOBULIN 21 TO 160 C.C.		
	CASES OF ENCEPHALITIS	SEQUELAE	DEATHS	CASES OF ENCEPHALITIS	SEQUELAE	DEATHS	CASES OF ENCEPHALITIS	SEQUELAE	DEATHS
0-4									
Number	22	2	4	5	0	2	5	0	1
Per cent	100	9.1	18.2	100	0	40.0	100	0	20.0
5-9									
Number	42	6	6	13	2	3	13	2	1
Per cent	100	14.3	14.3	100	15.4	23.1	100	15.4	7.7
10-14									
Number	3	0	0	3	1	0	6	1	0
Per cent	100	0	0	100	33.3	0	100	16.7	0
Total									
Number	67	8	10	21	3	5	24	3	2
Per cent	100	11.9	14.9	100	14.3	23.8	100	12.5	8.3

conclusion is inevitable that gamma globulin is of no value in the treatment of measles encephalitis.

SUMMARY

1. Over a period of five and one-half years there were observed 159 patients with measles encephalitis under the age of 15 years. Of these, 51 were treated with gamma globulin and 108 were not.

2. About 25 per cent of the children died or developed permanent sequelae, and the percentage was the same in the two groups.

3. No significant differences in results between the treated and the untreated were found when comparisons were made by age groups or by amount of treatment or by severity of case, irrespective of whether the difference was based on deaths or sequelae or both.

4. Gamma globulin has no value in the treatment of measles encephalitis.

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PSEUDOMEMBRANOUS ENTEROCOLITIS IN INFANCY

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RECENTLY, there has been a resurgence of interest in pseudo membranous enterocolitis. This condition, so named for its histopathologic characteristics, consists of an inflammation of the gastrointestinal tract associated with mucosal ulceration and the formation of a pseudomembrane. It is considered to be of serious prognosis and often terminates in death, though a recent publication indicates that successful medical management is possible.

A number of articles have appeared in the literature dealing with the increased recognition of the entity in adult patients but little has been written concerning the occurrence in patients of the pediatric age range. Because of the paucity of data concerning this entity in infancy and childhood and because our own experience has revealed an extremely grave prognosis to be associated, we have undertaken the following study.

METHODS

The protocols from 1,333 consecutive autopsies were reviewed and all cases having functional or mechanical intestinal obstruction, inflammation of the gastrointestinal tract, or peritonitis were collected. This material represents the entire autopsy experience of the Children's Hospital from January, 1942, through September, 1954. All available microscopic sections of the gastrointestinal tract from the cases in the above categories were carefully examined and those filling the histopathologic criteria for the diagnosis of pseudomembranous enterocolitis form the basis of this study.

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SURVEY OF CLINICAL RECORDS

Ten cases of pseudomembranous enterocolitis were found in the material cited. The clinical records of the ten were carefully reviewed in conjunction with the autopsy protocols and microscopic sections.

The ages varied from 10 days to 9 months and there was an 8:2 ratio in favor of males. Nine of the ten patients were of the Caucasian race and one was Negro. This race distribution approximates our general admission ratio.

The signs which appear to be related to the condition of pseudomembranous enterocolitis in the pediatric age group are quite similar to those mentioned by investigators dealing with adult patients. The case records indicate that this process, once instituted, is of extremely grave prognosis and generally of very short duration. Though the initial illness in the ten cases varied, the acute signs relating to the colitis developed twenty-four to seventy-two hours before death. Table I lists the more constant of the signs seen in the ten cases.

The patients who had a febrile reaction associated with their illness generally demonstrated this sign in rather marked degree. The temperatures varied from 99.2° F. to 107° F. rectally, with an average of over 103° F.

TABLE I. SIGNS ASSOCIATED WITH PSEUDOMEMBRANOUS ENTEROCOLITIS IN INFANCY

Distention		9
Fever		8
Shock		8
Vomiting		7
Diarrhea		6
With blood	3	
Without blood	3	
Carpopedal spasm		4

The abdominal distention was the most prominent and constant of the signs recorded on the charts and was in most instances thought to be quite marked.

The significant similarities of laboratory findings were limited to the white blood cell counts. Three of the patients had a white blood cell count of over 20,000 per cubic millimeter and the range was from 6,200 to 46,000 per cubic millimeter. The differential counts were, in general, within normal range of distribution of elements.

Stool cultures revealed normal flora in all but two cases in which staphylococci were found.

Four of the infants had received antibiotics previous to the onset of the characteristic signs and the antibiotics involved were Terramycin, penicillin, streptomycin, Achromycin, erythromycin, and sulfonamides.

Two patients had undergone surgical procedures prior to the onset of their signs. In one there was an ileostomy and reduction of an intussusception, and in the other there was a colectomy. The latter was done three weeks prior to the final illness, and the former twenty-four hours prior to death.

In order to emphasize the severity of the prognosis and the clinical course, a brief summary of one of the cases is included.

CASE REPORT

C. H. (Case No. 53-75187).—This white male infant was born on Oct. 3, 1953, and was the product of a normal, full-term pregnancy. The labor and delivery were not unusual, and the birth weight was 7 pounds 13 ounces. At the age of 3 days, he vomited some mucus and greenish fluid and became distended. The vomiting increased and he was said to have passed several meconium stools just prior to admission.

On admission to Children's Hospital (Oct. 7, 1953) his temperature was 98.4° F. and the physical examination revealed little save for abdominal distention and the absence of bowel sounds.

The hemogram disclosed a normal red blood count and hemoglobin, with a white blood count of 11,700 per cubic millimeter. The CO_2 capacity of the serum was 18.2 meq. per liter and the serum chlorides were 97.7 meq. per liter. A flat plate of the abdomen revealed stomach, small bowel, and colon distended with gas. Wangensteen suction was instituted for several hours and the distention receded somewhat. This was discontinued later on the first day and formula was given. The infant seemed to do fairly well. There was no diarrhea and only a small amount of vomiting. There was a slight weight gain. On October 10 a barium enema revealed what was thought to be a normal colon. On October 14 an abdominal flat plate revealed a narrow descending colon still filled with barium and the remainder of the gastrointestinal tract was somewhat less distended. On this same day, the

baby began to have increased amounts of vomiting and the examiner felt that the infant showed evidence of pain associated with peristaltic rushes. Wangensteen suction was again instituted. During the remainder of the day the infant became more and more distended, and on the following morning (October 15) he was noted to be distended, toxic, and acutely ill. It was at this point that he began to have convulsive twitching with marked carpopedal spasm. Calcium was seen to relieve this when phenobarbital had no effect.

From this point, the infant's condition rapidly deteriorated, the temperature elevation reached 103° F., abdominal distention became marked, and respiratory weakness developed. The following morning (October 16) the infant was cold, cyanotic, and in obvious shock. Emergency supportive measures were instituted and penicillin and streptomycin therapy was started, but, despite all efforts, the patient died. An autopsy was granted.

At autopsy, the body was that of a well-developed and fairly well-nourished, white male newborn infant. The skin was slightly cyanotic and the abdomen was moderately distended.

The heart, lungs, spleen, kidneys, and adrenals were within normal limits.

The abdominal cavity contained 100 c.c of yellow fluid and the small bowel was hemorrhagic. There were flecks of yellowish fibrinous material attached to the serosa. The ascending colon was markedly dilated. The arteriovenous pattern of the gastrointestinal tract was unremarkable. The mesenteric lymph nodes were red, succulent, and bulging. They measured up to 1 cm. in diameter.

Discoloration of the small bowel was noted, beginning 40 cm. distal to the ligament of Treitz and extending to the ileocecal valve. The bowel proximal to the involved area had a circumference of 3 cm. and its wall measured 1 mm. in thickness. The involved small bowel had a circumference of 5.5 cm. Its mucosa was markedly hemorrhagic and there was a thick, yellowish membrane over its surface. Stripping this membrane away left a raw and hemorrhagic surface. The membrane averaged 2 mm. and the wall from 2 to 4 mm. in thickness. In a few areas, the membrane formed a complete rubbery yellow cast of the small bowel lumen.

Similar membrane was seen, in a patchy distribution, on the mucosa of the cecum and ascending colon to a point of sharp constriction adjacent to the gall bladder. The colon proximal to this constriction measured 7.5 cm. in circumference and had a wall 3 mm. in thickness. Distal to the constriction, the colon circumference was 3 cm. and the mucosa was unremarkable.

The final diagnosis was (1) congenital megacolon (Hirschsprung's disease) with absence of ganglion cells in the plexuses of Auerbach in the spastic segment; (2) pseudomembranous enterocolitis; (3) transmigration peritonitis; (4) subacute mesenteric lymphadenitis.

PATHOLOGY

The term enterocolitis, which is applied to this condition, arises from the fact that the small bowel and colon are the sites of most common involvement but our series revealed that the remainder of the gastrointestinal tract is also at times affected. Table

TABLE II. BOWEL SEGMENTS SHOWING PSEUDOMEMBRANOUS LESIONS

Esophagus	2
Stomach	2
Duodenum	4
Jejunum	5
Ileum	6
Cecum	7
Ascending colon	7
Transverse colon	5
Descending colon	6
Sigmoid colon	5
Rectum	2

II lists the segments of the gastrointestinal tract involved in the ten cases and tabulates the number of cases in which each segment was affected.

In the five cases which were associated with intestinal obstruction, it was noted that the gross lesions of the gastrointestinal tract were never found distal to the point of obstruction.

In four cases, swelling and redness of the mesenteric lymph nodes were noted. In an equal number, fibrin and increased amounts of fluid were found in the abdominal cavity.

result of interaction of concomitant primary or secondary diseases.

A vascular change is the first alteration noted. This is composed of general dilation of the arterioles, venules, and capillaries. The change is most marked in the latter two and especially in the lamina propria and submucosa. Engorgement is severe in these dilated vessels. Capillary hemorrhages may be prominent in the lamina propria especially in the tips of the mucosal folds (Figs. 1, 2, 3).

Edema of the submucosa and subserosa seems to follow the initial

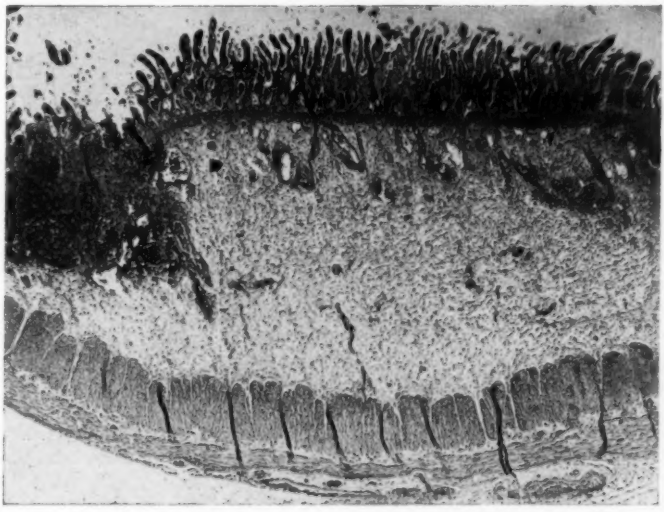

Fig. 1.—Early lesion showing mucosal and submucosal congestion and submucosal edema. (×23.)

The histopathologic development of the mature lesion of pseudomembranous enterocolitis appears to follow a rather standard pattern and may arbitrarily be broken down into several phases. These are in general agreement with the descriptions of other authors. Variations occur as the

vascular change (Figs. 1, 2, 3). Tissue macrophages in the edematous areas are prominent. Following or accompanying the edema, there is a sparse transmigration of leukocytes from adjacent vessels. Along with this second phase, changes in the mucosal epithelium are detected. Cessation of

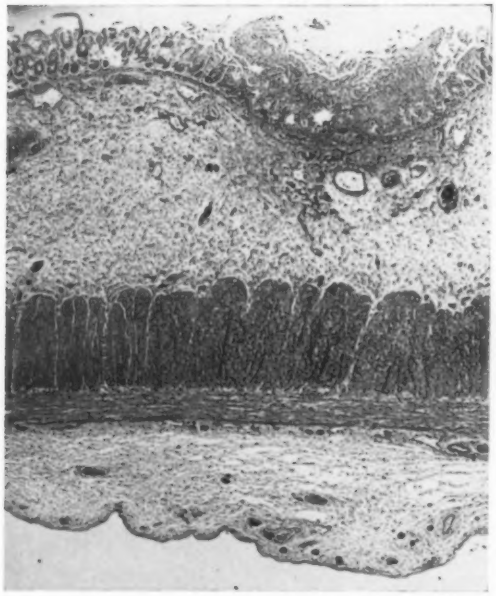

Fig. 2.—Intermediate lesion showing early pseudomembrane formation and marked edema of subserosa and submucosa. (×23.)

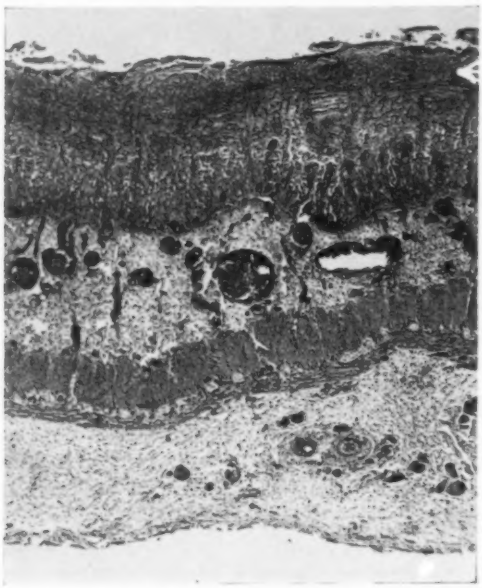

Fig. 3.—Advanced lesion. Note marked congestion of the submucosa. (×23.)

mucous production is followed by hyperchromatism of the nuclei and a loss of basal nuclear position. Pyknosis of the nuclei is noted, especially in the tips of the mucosal folds, and this is followed by epithelial sloughing. As the epithelial degeneration proceeds, fibrin deposition is noted in the lamina propria (Fig. 2). In some areas, fibrin thrombi are seen in the laminar capillaries (Fig. 4). The fibrin, accompanied by leukocytes, soon forms a membrane over the denuded mucosa which is completed by a luminal layer of degenerated leukocytes, fibrin, and at times bacteria (Fig. 3).

DISCUSSION

Certain inflammatory conditions of the gastrointestinal tract, associated with the formation of a pseudomembrane, are of known origin. It is well recognized, for instance, that the lesions produced by some of the specific dysenteries, heavy metal poisoning, and uremia are accompanied by the formation of pseudomembranes.

The cases which make up this series fall into a group for which no specific etiology has been demonstrated. Contemporary investigators dealing with pseudomembranous enterocolitis of nonspecific origin have pointed out a

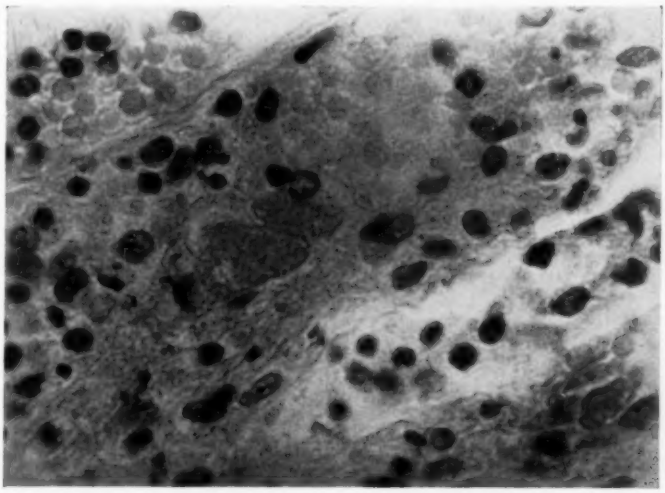

Fig. 4.—Note fibrin thrombus in a laminar capillary. (×516.)

The muscularis is congested and focally hemorrhagic. Although the subserosa is edematous and macrophages are present, a frank, acute, purulent peritonitis is unusual unless stimulated by some other motivating entity such as intussusception.

number of conditions which frequently are associated with it. On the basis of the frequency of appearance, suggestions of possible etiological significance have been advanced. Table III indicates the relation of our experience to the sug-

gested associated entities mentioned by previous authors. These are: *Staphylococcal enteritis,*[1-3, 5, 6, 10, 11, 16, 17] *surgical operations,*[4, 7, 14] *intestinal obstructions,*[14] *antibiotic therapy,*[15] *shock,*[12, 13] and *nonstaphylococcal sepsis.*[14]

As noted in Table III, shock was associated with the illness in eight of the ten cases. However, in all instances, it was a terminal phenomenon and for this reason no etiological significance can be attached to it. This conclusion is not in agreement with certain other investigators.[12, 13]

TABLE III. FACTORS ASSOCIATED WITH PSEUDOMEMBRANOUS ENTEROCOLITIS

Shock	8
Intestinal obstruction	5
Antibiotic therapy	4
Sepsis (nonstaphylococcal)	3
Surgical operations	2
Staphylococcal enteritis	2

Of the intestinal obstructions noted, two were congenital megacolon, one a congenital diaphragm, one a pyloric stenosis, and one an intussusception. Of all the factors mentioned, the intestinal obstructions seem to be the most closely related. This is in keeping with the observations of Pettet and associates[14] who found obstructing carcinomas of the colon in a large number of their cases of pseudomembranous enterocolitis. In all of our cases, the obstructions were present before the signs of pseudomembranous enterocolitis had appeared, and it is of perhaps more significance that the lesions noted in these cases were never found distal to the point of obstruction.

Although the bulk of the literature on pseudomembranous enterocolitis portrays staphylococcal enteritis as an important factor in the production of the lesion, only two of our cases were proved to have this organism in the stools.

Antibiotic therapy was not significantly related to the origin of the pseudomembranous lesions in our cases.

The existing literature points out that the factors discussed are associated with the disease and are of etiological significance. Our material corroborated the association of the factors mentioned, but fails to reveal specific etiological relationship.

The diagnosis of the development of pseudomembranous enterocolitis appears to lie entirely within the realm of clinical appraisal. X-ray examination, either with or without contrast media, has not helped in establishing the diagnosis in our series of cases. However, Dr. R. P. Allen of the Children's Hospital Roentgenology Department was able to correctly suggest the diagnosis of one patient not hospitalized in our hospital. In this case, the barium-filled bowel revealed a peculiar layering which was interpreted as being produced by barium dissection of the pseudomembrane. Perhaps further investigation of this phenomenon in later cases may prove of value.

Of the clinical signs, the frequently appearing carpopedal spasm is perhaps the most thought-provoking. This sign is one which certainly cannot be considered a common one in infancy other than when related to Snelling's syndrome, parathyroid disease, or rickets. No calcium levels or calcium ion concentrations were done on any of the ten patients, but it is perhaps of significance that in one case the carpopedal spasm was resistant to phenobarbital and re-

sponded quickly to the intravenous injection of calcium gluconate. Three of the four patients who demonstrated this sign had moderate to severe vomiting, Wangensteen suction, or both. It is plausible that the loss of chloride with the gastric secretions could have been the inciting factor. At least it is conceded that such loss can produce metabolic alkalosis and result in the reduction of the serum calcium ion concentration. The origin of this sign should certainly be defined more accurately in future cases.

It is obvious that this condition presents a challenge to the clinician from the standpoint of early diagnosis and therapy. Knowledge of the existence of this disease and a high degree of suspicion when cases of the types illustrated are encountered will undoubtedly be necessary if diagnosis is to be made in time for therapy to be of value. The pediatric surgeon will at once recognize that the signs of pseudomembranous enterocolitis are similar to those of other complications seen in cases of intestinal obstruction or postoperative surgical problems. Although peritonitis, perforation, and leakage of an anastomosis will undoubtedly be more common, pseudomembranous enterocolitis should be considered in the differential diagnosis.

From a therapeutic standpoint, several factors are evidently of great importance. Perhaps the most important is the time element, as infants developing this condition die within twenty-four to seventy-two hours after the onset of the recognizable signs. Since shock plays such an important role in the exitus of the patients, as evidenced by the ten cases reported, recognition of this state would seem less important than its anticipation.

Cultures of the stools or of rectal swabs should be taken, and smears of the latter examined for the presence of organisms morphologically resembling staphylococci. Although sensitivity tests should be made, therapy is not to be withheld in such cases while awaiting the results. The presence of organisms in the smears morphologically similar to staphylococci is indication enough for the use of erythromycin. The absence of these organisms does not negate the diagnosis of pseudomembranous enterocolitis. This is illustrated by our series in which only two of ten patients had this valuable sign.

Once the case is recognized, means of rapidly administering intravenous fluids, blood, and plasma should be instituted. Careful attention to electrolyte and fluid deficits and their rapid replacement is essential. The use of calcium gluconate on a prophylactic basis is perhaps warranted. Corticotrophin has been suggested on the basis of the possibility that the terminal shock is a result of adrenal failure. Nyhus and Burke[11] employed hydrocortisone 200 mg. intravenously initially and 200 mg. intramuscularly daily in the successful treatment of a 9-year-old boy ill with the disease. We feel that the gastrointestinal tract should be put at rest with the use of Wangensteen or Miller-Abbott drainage.

The case reported by Nyhus and Burke and other instances of successful therapy which they quote prove that the condition of pseudomembranous enterocolitis cannot only be recognized but can be treated by

medical means with good results. It is to be hoped that this can be accomplished for the infant as well as for the older child, in spite of the smaller "margin for error" characteristic of the treatment and care of the former.

SUMMARY

Ten cases of pseudomembranous enterocolitis are presented which represent twelve years of autopsy experience at the Denver Children's Hospital.

The clinical and pathologic findings of the ten cases are analyzed and a discussion of etiological and associated factors is presented.

An approach to the diagnosis and therapy of this condition in infancy is suggested on the basis of facts revealed by this study and the experience of other authors.

We wish to express our appreciation for the advice and assistance of Dr. H. D. Palmer and for the photographs by Mr. I. S. Stephens, M.T.

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FAMILIAL DYSAUTONOMIA

Case Report With Autopsy

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IN 1949 Riley and co-workers[1] reported five cases of autonomic dysfunction with defective lacrimation. The case we are reporting meets the diagnostic criteria for this disease, and is in fact mentioned in Riley's latest series of twenty-eight additional cases.[2]

Although the clinical features of the disease have been well described, post-mortem findings have been meager. Autopsies have been performed in nine previous cases, and central nervous system findings have been noted in only two cases. In one patient, lesions probably unrelated to the syndrome (multiple brain abscesses secondary to aspiration pneumonia) were noted. The other patient reported by Engel and Aring[3] in 1945 had a typical clinical picture and at autopsy showed a cyst which destroyed the ventral half of the dorsomedial nucleus, a bit of the internal medullary lamina and of the lateral nucleus of the thalamus on the right. There was also unilateral rarefaction of the reticular substance in the pontine, medullary, and upper cervical regions. The hypothalamus was intact.

We are reporting this case as a second instance of pertinent neuropathological findings. We feel that these lesions are significant and may shed light on the pathogenesis of this unusual disease.

From the pediatric service of Beth Israel Hospital.

CASE REPORT

First Admission.—A. H., a Jewish male infant aged 9 months, was first admitted to Beth Israel Hospital in July, 1949, with a history of fever of seven days' duration, ranging from 103° to 104° F., cough, and wheezing respiration.

The history revealed a normal spontaneous full-term delivery. The birth weight was 6 pounds 4 ounces. The infant had taken food poorly and gained slowly. At 7 months he developed "virus" pneumonia and at 8 months, eczema.

The mother had had one previous child who lived but six hours. No autopsy was performed.

Physical examination revealed the following findings: temperature 100° F., pulse 100, respirations 30. The child was thin and did not appear acutely ill. Eczema was present on face, abdomen, and arms. A few râles were heard scattered over both lung fields. The feet were held in pes cavus position. Neurological examination revealed deep tendon reflexes equal in the upper extremities but not elicited in the lower extremities. Decreased muscle tone was noted in the legs.

Urine was normal. Red blood count was 4.05, hemoglobin 10.5 grams, white blood count 10,750, polymorphonuclears 62 per cent.

Chest roentgenogram revealed no abnormality of the cardiac silhouette or pulmonary fields.

The child was treated with sulfadiazine and responded promptly. He was discharged on the eleventh hospital day.

Second Admission.—The patient was readmitted at 15 months of age be-

cause of fever of unknown origin. Since the previous admission the child had run an irregular fever ranging up to 102° F. associated with a slight nonproductive cough. The child sat with help at 10 months and his first tooth appeared at that time. He was unable to stand at 15 months.

Previous examination and laboratory findings were similar to those of the previous admission except that at this time a roentgenogram of the chest showed increased peribronchial markings throughout both lungs, and patchy infiltrations in the right lower lung region. Roentgenograms of the long bones of the lower extremities revealed slight osteoporosis. Skull and spine roentgenograms were not remarkable. Muscle testing showed no evidence of reaction of degeneration. I.Q. was reported as 63.

The child ran an irregular fever in spite of Chloromycetin, Aureomycin, and streptomycin medication. On the twelfth hospital day tarry stools were noted which were benzidine-positive for blood. Gastric contents were also positive for blood. Tarry stools continued for four days; roentgenograms of the gastrointestinal tract revealed no lesions. The child was discharged after three weeks of hospitalization.

In the ensuing fifteen months, the patient had five additional respiratory episodes of bronchitis and pneumonitis which were treated at home with Chloromycetin and Terramycin.

Third Admission.—The patient was admitted for the third time at 2½ years of age with a one-week history of cough, fever, anorexia, and vomiting. Additional history at this time revealed that the child did not stand until 2 years of age and did not walk until 28 months. At 2½ years of age the child spoke only a few words although he seemed to understand commands well. The mother stated that the child perspired profusely, had cold hands and feet, and cried without tears. Skin blotching was noted when he cried. During temper tantrums for one year previous to admission, he became "stiff" for a few seconds.

Physical examination revealed temperature 105° F., pulse 120, respiration 70, blood pressure 110/68. This examination was similar to that of the first admission.

Urine specific gravity ranged from 1004 to 1030. Bleeding and coagulation times were normal. Blood glucose, nonprotein nitrogen, and total protein were normal. The blood electrolyte studies showed the following (reported in milliequivalents per liter). Sodium 141, potassium 3.6, chloride 97.1, carbon dioxide 18.7. Electrocardiogram was normal. Electroencephalogram was interpreted as normal.

For the next six weeks the child ran an irregular temperature up to 105° F. which did not respond to Chloromycetin, streptomycin, or Terramycin. Frequent blood pressure determinations during this period showed occasional readings of increased systolic and diastolic pressures, as high as 170/110. Most readings, however, were between 110 and 130 systolic and 68 to 80 diastolic. The child's temperature returned to normal during the sixth week of hospitalization.

Fourth Admission.—The patient was admitted for the fourth time 2½ months later with a history of fever, convulsions, and hematemesis of five days' duration.

Physical examination was essentially unchanged. The blood pressure was 100/50.

Laboratory data: Nonprotein nitrogen was 165 mg. per cent, sodium 126 Meq. per liter, carbon dioxide 9.8 Meq. per liter. Intravenous pyelogram showed no abnormalities: phenolsulfonphthalein test was normal. Electroencephalogram and gastrointestinal series were repeated and reported as normal. Chest roentgenogram was unchanged.

The child was given parenteral fluids, whole blood, and streptomycin. Improvement was noted the following day. Vomiting ceased although tarry stools still persisted. Nonprotein nitrogen was 125, 60, and 19 mg. per cent on successive days. Potassium, sodium, and carbon dioxide rose to

normal within the next few days. On the fifth hospital day oral feedings of milk and cream through a polyethylene catheter were started. He continued to run a low-grade fever despite courses of streptomycin, Chloromycetin, Aureomycin, penicillin, and phenobarbital. Blood pressure 130/80 was unchanged by cold pressor test, whereas pinprick resulted in blood pressure of 160/90.

Fifth, Sixth, and Seventh Admissions.—There were two subsequent admissions within the next six months for recurrent episodes of hematemesis, which were treated in a similar manner.

The patient's final admission was in June, 1952, at 3½ years of age, again with hematemesis of thirty-six hours' duration.

The temperature was 106° F., blood pressure 100/60, pulse 140, respirations 24. Other physical findings were as noted on previous admission.

On the day following admission the patient suddenly died.

Post-mortem examination was done by the Pathological Department of Beth Israel Hospital. With the exception of the nervous system, there were few abnormalities noted at autopsy. Pulmonary emphysema and focal atelectasis were noted in both lungs. Right testicle was located in the inguinal canal. Left testicle was not seen either in the scrotum or inguinal canal. The kidneys weighed 35 grams each and showed no gross abnormalities. Microscopically, the usual architecture was present. There were numerous dark-staining masses in the tubules and calcific casts. There was also some light pink-staining albuminous material in many of the convoluted tubules. The blood vessels were engorged. The esophagus was normal except for a pink area (1.5 by 3.0 cm.) elevated 0.2 cm. above the surrounding mucosa located on the posterior wall of the esophagus 2 cm. above the gastroesophageal junction. Section of the esophagus and paraesophageal nodes showed dilated blood vessels in the paraesophageal tissue.

The neuropathological examination was made by Dr. H. M. Zimmerman. The following is his report:

Neuropathology, Gross.—The brain was well developed, the convolutions revealing their usual tortuosity. The pons and medulla were smaller than expected, resulting in undue prominence of the inferior olives. The cerebellum was of average size. The ventral vermis seemed to be slightly decreased in size, allowing the fourth ventricle to lie exposed because of the incomplete formation of the roof of this cavity. On cross-section of the midbrain there was nonpigmentation of the substantia nigra. At the junction of the midbrain and pons the reticular substance appeared to be atrophied.

Fig. 1.—Transverse section of pons to show enlarged fourth ventricle and atrophic reticular formation.

The spinal ganglia were of average size. The cauda equina appeared to be atrophied.

Neuropathology, Microscopic.—Cortical structures revealed an essentially normal pattern. The neurones of the substantia nigra were well preserved

but lacked the melanin pigment of older cells. (For a 4-year-old child this condition is quite normal.)

Swollen myelinated fibers and myelin fragments are found in the reticular substance of the pons (Fig. 1). There is phagocytosis of lipid material by macrophages.

The most extensive lesion lay within the dorsal half of the medulla and was seen best in the preparation of this structure which included the tenth and eleventh cranial nerve nuclei. The neurones of these nuclei were shrunken and possibly even reduced in number. but the lesion involved the nerve fibers

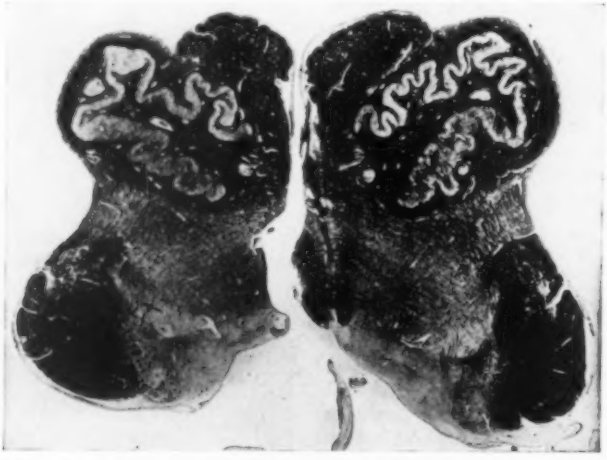

Fig. 2.—Photomicrograph of medulla. The myelinated fibers of the reticular formation are sparse. (Kultschitzky stain for myelin.)

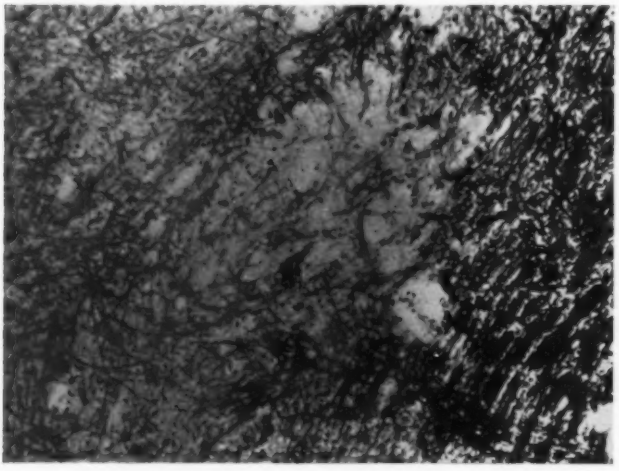

Fig. 3.—The myelin sheaths of the reticular formation in the medulla are fragmented and form balloons. Many have completely degenerated. (Kultschitzky stain for myelin.)

of the reticular formation much more than the cells (Fig. 2). The nerve fibers were extensively damaged; they were irregularly swollen and ribbon-like; they were fragmented and formed many balloons (Fig. 3). A moderate phagocytic reaction occurred in which monocytes were seen filled with fatty material. In the ventral half of the medulla, the inferior olives and the pyramids appeared to be intact. The medullary lesion was not associated with any inflammatory reaction in the nature of an encephalitis, nor did it appear to be the result of vascular occlusion.

Sensory as well as motor tracts in the spinal cord were well myelinated. The central gray matter appeared somewhat atrophic, but it was difficult to be certain of a genuine loss of ganglion cells. The dorsal as well as ventral nerve roots were perfectly myelinated.

Most of the cerebellar folia were well preserved, but both in the lateral cerebellar lobes as well as in the vermis there were some folia which were partially and even completely denuded of Purkinje cells. This loss of neurones, more marked in the vermis, was unassociated with gliosis or evidence of inflammation. There are no destructive lesions in the medullary fibers.

Summary of Findings.—The chief or outstanding pathologic changes in this case were confined to the reticular formation of the lower pons and especially the medulla. They affected the myelin sheaths primarily but also involved several of the cranial nerve nuclei in the floor of the fourth ventricle. They resulted in some atrophy of the restiform bodies and were associated with either an agenesis or a destruction of certain Purkinje cells in the cerebellum. The nature of the lesion could be described as "degenerative" which, however, gives no clue as to its etiology.

Diagnosis.—Destruction of reticular formation of pons and medulla.

DISCUSSION

The clinical features of this disease have been conveniently divided by Riley[2] into two groups: (A) those almost always present and (B) those occurring frequently enough to make them appear relevant (Table I).

TABLE I

Group A	Group B
1. Jewish extraction	1. Hypertension
2. Defective lacrimation	2. Cyclic vomiting
3. Skin blotching	3. Frequent pulmonary infection
4. Excessive perspiration	4. Frequent unexplained fever
5. Drooling	5. Breath-holding spells in infancy
6. Emotional instability	6. Urinary frequency
7. Motor incoordination	7. Mental retardation
8. Hyporeflexia	8. Convulsions
9. Relative indifference to pain	9. Corneal ulceration

Our patient showed all the features of group A and all but one (corneal ulceration) of group B.

In addition, our patient had pes cavus and several episodes of hematemesis. These features may also be part of this clinical syndrome. Skin blotching usually appeared on the face and upper thorax when the patient was excited. In addition, at the age of 6 months he had an exudative dermatitis which had the typical appearance, distribution, and course of atopic eczema.

As pointed out by Riley, the child's relative indifference to pain was well demonstrated by his diminished reaction during the cold pressor test.

Hypertension, which was noted at times to be as high as 170/110 mm. Hg, was neither prominent nor specifically related to his frequent vomiting episodes. This was in contrast to another case of this disease seen at Beth Israel Hospital in which the hypertensive episodes simulated pheochromocytoma.[4]

Cyclic vomiting (and, later hematemesis) was the outstanding and most troublesome manifestation in our case. During one episode azotemia (165 mg. per cent nonprotein nitrogen) was present, which, with adequate parenteral therapy, receded to normal in five days.

Pulmonary findings on auscultation and roentgenographic evidence of pulmonary infiltration accompanied the recurrent febrile episodes. The pulmonary picture simulated mucoviscidosis, but tests for this disease were negative.

Three convulsions were noted in our case and each time they occurred after the sudden onset of fever to 104° F. or higher. We felt that they might best be classified as febrile convulsions.

Having had the opportunity to follow our patient from birth to death, we think the following clinical features are important in the early diagnosis of this disease.

1. Difficulty in feeding and failure to gain weight.
2. Delayed motor development.
3. Defective lacrimation.
4. Hypoactive to absent knee jerks.
5. Skin blotching.
6. Excessive perspiration.

Prognosis in this disease is poor. Approximately one-third of thirty-three patients have died during childhood, although one of Riley's patients is almost 20 years of age.

Treatment has been singularly unsuccessful. Autonomic muscle relaxants, cortisone, tentorial splitting, lumbar sympathectomy, prefrontal lobotomy, psychotherapy, and other measures have all failed to produce any lasting benefit.

It will be noted that while some of the clinical features of this disease are associated with parasympathetic stimulation or sympathetic inhibition (perspiration, drooling, and skin blotching), other signs are characteristic of sympathetic stimulation or parasympathetic inhibition (defective lacrimation, hyperthermia, and hypertension).

Autonomic regulation, although influenced by mechanisms at all levels of the central nervous system, is believed to have its main center in the hypothalamus. Two functionally different regions are recognized: (a) anterior and midline nuclei which are concerned with parasympathetic outflow, and (b) posterior and lateral nuclei, stimulation of which results in sympathetic response.

Magoun[5] has described a pathway in cats which arises in the lateral hypothalamic area and descends to the preganglionic sympathetic neurones in the spinal cord. In the medulla this tract is chiefly situated in the lateral reticular formation. List and Peet[6] concluded that in man descending hypothalamic tracts were situated in the same anatomic areas.

In our case, the principal pathologic change in the central nervous system was a destruction of the reticular formation of the pons and medulla. The hypothalamic centers were relatively intact. Destruction of the centers would be expected to produce a fixed syndrome of autonomic dysfunction. A lesion involving the hypothalamic tracts would also explain the

varied and paroxysmal clinical features of this disease. Perhaps there is an imbalance in autonomic function facilitated by the lesions in the hypothalamic pathways.

Bodian,[7] in studying the histopathology of poliomyelitis, found that lesions in the central gray matter of the midbrain and the hindbrain and in the midbrain tegmentum and reticular formation were very common, and often severe. Extensive involvement of the reticular formation has often been recorded. Some cases without bulbar signs showed severe lesions in this region. Recent reports have noted not only respiratory and swallowing difficulties but also hypertension and gastrointestinal disturbances, sometimes with hemorrhage, ulcer formations, or perforation. Disturbances of respiratory, vasomotor, and motor inhibitory mechanisms (spasm of muscles) are correlated in many instances with extensive lesions in the reticular formation.

Vomiting and hematemesis were prominent clinical features in our case. Approximately two-thirds of Riley's series had severe attacks of vomiting. Watts and Fulton[8] demonstrated in monkeys that lesions confined to the tuberal nuclei may lead to hematemesis and multiple erosions of the gastric mucosa.

Welt and associates[9] have shown an influence of autonomic brain centers and pathways on water and electrolyte metabolism. Lesions result in tremendous deficits, requiring immediate intensive intravenous fluid and electrolyte replacement. Characteristic is elevation of blood nonprotein nitrogen to high levels. This has also been our experience in cases of familial autonomic dysfunction with intractable vomiting. In those patients who died, renal pathology was absent so that death was due not to uremia but rather to this critical disturbance of water and salt metabolism.

Excessive perspiration, sometimes asymmetrical, is a constant feature of familial autonomic dysfunction. Sweating disturbances were described by List and Peet in patients with lesions of the pons, medulla, and cervical region of the cord. The descending sweat fibers pass through the tegmental portions of the pons and the lateral reticular gray matter of the medulla. Thus the lesions described in our case could produce the deviations in sweat secretions so often seen in this disease.

Concerning the etiology of this disease, the lesions in the nervous system found at autopsy were degenerative rather than inflammatory. Furthermore, Riley found that one-third of all known siblings of the index cases were thought to be affected. He postulated that this disease is familial and is transmitted as a mendelian recessive. Thus we seem to be dealing with a familial degenerative disease of the central nervous system predominantly affecting hypothalamic pathways and producing a syndrome of autonomic dysfunction.

Variation of extent of pathological findings may account for the variation in the symptomatology of the disease. We need not have the complete syndrome in all cases.

CONCLUSION

1. A case of familial autonomic dysfunction with autopsy findings is presented.

2. The clinical features of this disease are reviewed.

3. Central nervous system lesions consisting mainly of degeneration of the reticular formation of the pons and medulla were found post mortem.

4. Since this area includes hypothalamic pathways, it may account for many of the clinical features of this disease.

5. Other findings in the cerebellum, cerebral cortex, and spinal cord may explain other features of this syndrome.

We are greatly indebted to Dr. Henry Zimmerman for his painstaking and detailed studies of the neuropathology of this case and also would like to express our appreciation of the aid given us by the pathology department of Beth Israel Hospital.

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On Position in Bed

Some use to lie their Children in the Cradles higher at their feet, than at the head or heart, at which I marvailed many times. But if a Child hath scabbed swelled leggs or feet, then their feet must be laid somewhat higher, that the blood run not too much into the feet, and the pulling itching pains be not increased thereby: let every one be judge here, that is troubled with naughty leggs, if he hath but a little blister about his foot or ancle, what pains he feeleth then, if his legg hangeth down: what do you think a Child endureth in this case.

If the Child be sound in body, leggs and feet, then it may lie higher at his feet than at his sides, but not higher than it lyeth at his heart and head: this I hold to be very good. Therefore my advice is, to make that part of the bed somewhat higher where the child's side is to lye, then his head and heart will lye higher than his feet and body, which is the safest and easiest way for his rest.

FELIX WURTZ (1518-1574)

THE USE OF PARTIAL VITAMIN SUPPLEMENTS IN THE TREATMENT OF GROWTH FAILURE IN CHILDREN

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MALNUTRITION has been recognized frequently in so-called normal children of school age, and as a result of acute and chronic illness. Indeed, the management of convalescence is, in part, a problem of assuring adequate calories to cover, in the child, both reparative and growth requirements. The existence of specific vitamin deficiencies, other than the full-blown avitaminoses, has not received extensive support. The most recent work suggesting a subclinical vitamin deficiency state has been that of Wetzel and associates[1, 2] and Chow[3] in their work with vitamin B_{12}. Wetzel found that a daily dose of 10 μg of vitamin B_{12}, when administered to children with retarded growth, promoted appetite and increased caloric intake sufficiently to produce growth responses in an appreciable percentage of children. His cases of simple growth failure were selected from a school population by means of Wetzel Grids. Chow has reported that a B_{12} supplement at 25 μg a day, orally, will increase the weight gains in institutionalized children suffering from chronic illnesses such as rheumatic heart disease, simple malnutrition, or chronic infection. Although other workers have reported negative results with B_{12} supplements, these studies are subject to the criticism that adequate selection of patients was not exercised in the design of the experiments.

If the studies of Wetzel and Chow are substantiated, then the administration of B_{12} alone or in combination with other members of the B group should aid in correcting either primary or secondary growth failure.

So far as subclinical B-complex deficiencies are concerned, only that of thiamine has been supported to any extent by clinical or experimental evidence. This may be attributed to the fact that the risk of inadequate intake is greater with B_1 than other members of the B complex. It may be seen from figures supplied by Elvehjem[4] that with borderline diets this risk is twice as great with thiamine as it is with riboflavin, and about two and one-half times as great as it is with niacin. B_1 is the one member of this group directly connected with the total caloric intake, particularly that represented by carbohydrate.[5] Further, in the face of loss or inadequate intake, thiamine is not retained by the body as effectively as other members of the B group.

It is noteworthy that the earliest symptoms of B_1 or B_{12} deficiency are anorexia, growth retardation, and

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then loss in weight. This is in contrast to other vitamin deficiencies in which general inanition appears first and appetite is not diminshed until the later stages. It seemed to us that the empiric use of a mixture of B_{12} and B_1 in the treatment of growth failures, as encountered in ordinary clinical practice, should be investigated. Therefore, we administered a daily oral dose of 25 μg of vitamin B_{12} (cyanocobalamin) and 10 mg. of vitamin B_1 (thiamine hydrochloride) to approximately fifty children seen in private practice who gave a clinical impression of undernutrition. This report describes the response to this regimen.

METHODS AND MATERIALS

It is logical that results obtained after supplementing the diet with vitamin B_{12} would be more valid if the background of the subjects were known for at least a year prior to therapy. This control period would not only make changes more meaningful, but would also permit each patient to act as his own control. It is also necessary to follow the progress of a patient as long as possible after B_{12} has been added to his diet to determine whether improvement is slow to start or transitory.

In the majority of the children studied, a report of poor appetite and/or a clinical impression of retarded growth and development was prominent in the clinical picture. Two children who were overweight and one who was very tall were also included out of curiosity (nothing happened to alter the course of these children in this study). All were children between the ages of 3 and 12 years, who had been under observation for at least a year before treatment although a few of the height and weight measurements were taken for a shorter prior period. Many had been under the care of one of us (J. C.) since birth. The average period of observation before beginning the dietary supplement was 28.1 months.

Growth failure in most instances was secondary to chronic disease. Thirty-two children had allergic disorders, including hay fever, vasomotor rhinitis, asthma, eczema, or gastrointestinal allergy. A few had an associated endocrine deficiency. Other diagnoses included congenital heart disease and feeding and behavioral problems. Clinically, the condition of each child was considered to be in a stabilized state and the treatment in the pre-experiment period was the same as that during the supplementation with B_{12} and B_1. The regime of many children, of course, had included supplements of the oil-soluble and water-soluble vitamins but not in the "therapeutic" quantities given below. These supplements were continued during this study. The environment, so far as could be determined, was unchanged.

Each child in this group was followed at home. To each mother were given bottles of a flavored solution containing 25 μg of B_{12} and 10 mg. of B_1 per 5 c.c.* (This material was stable by microbiological assay for over two years. Total cobolamines determined chemically could be recovered after the same length of time.) Each mother was instructed to administer one teaspoonful of the

*This material was supplied by the Research and Development Division of Smith, Kline & French Laboratories, Philadelphia, and has since been marketed under the trademark Trophite.

supplement to her child daily. Except for the restrictions imposed by specific allergies, the diets at home were of free choice.

The heights and weights of the children were recorded at frequent intervals. Where prior observation was sufficiently complete to establish a child as his own control, his progress was plotted on Wetzel Grids. Adequate data were available for thirty-two children. The data were then subjected to the analysis reported below (R. T.). Each child was kept on the supplement for at least a year. Observations on many of the children were extended beyond the second year. The average period for the figures reported below was 15.7 months. Supplementation was initiated for most between January, 1950, and October, 1950. Seasonal spurts in growth were minimized by staggering the admissions to the study and by the length of observation following initiation of treatment.

RESULTS

Clinical Summary.—Parental reports of increases in appetite and improvement in activity and disposition were almost uniform. (This was in contrast with the experience encountered when the vehicle alone was used in approximately twelve patients.) Prior to therapy, appetite was considered to be normal in three, equivocally depressed in four, moderately depressed in ten, and markedly depressed in thirty-three. Nutrition by clinical estimate was regarded as normal in three, equivocally poor in twenty-two, and definitely poor in twenty-five. Quite early there was a definite impression that the use of this daily supplement of 25 μg of vitamin B_{12} and 10 mg. of vitamin B_1 was exerting a beneficial effect in the management of these children. Patient acceptance of the vitamin preparation was good and, as a result, the reliability of administration by the mother assured. On physical examination, improvement in many children was noted during the period of the study, and in many this seemed unexpected in view of their previous course. A summary of the height and weight changes of the individual children (Table I) supported the impression that in many children there were increases in physique apparently due to the administration of the two vitamins.

A. *Grid Analysis.*—Because of the heterogeneity of physiques in the ordinary child population, the estimation of undernutrition is difficult except in extreme cases. Evidence in physical examination is less accurate for the detection of growth failure in the individual child than the systematic plotting of the children's growth on a chart such as the Wetzel Grid. The lack of overt stigmas in subclinical deficiencies makes the use of continuous plots to detect both failure and recovery imperative. In addition, since the Wetzel Grid takes into account variations in growth rate correlated to chronological age, it is possible to avoid the all-too-frequent error of attributing an improvement to a therapeutic regime when it is actually due merely to time.

As noted, thirty-two cases were complete enough to permit analysis by this technique. In order to detect the effect of the nutritional supplement, it was necessary to classify these children by a method independent of the

TABLE I. CLINICAL SUMMARY OF FIFTY

CASE NO.	INITIALS	AGE AT B	SEX	DIAGNOSIS	REPORTED APPETITE −4 TO +4	ESTIMATED UNDER NUTRITION 0 TO −4
1	W. J.	6	♂	Twin, congenital heart	+1	+2
2	C. J.	6	♂	Twin, malnutrition	+1	+2
3	M. B.	7	♀	G.I. allergy	−3	+3
4	D. C.	8	♀	Feeding problem, behavior problem	−3	+4
5	J. K.	12	♀	Asthma, urticaria	−3	+3
6	G. K.	6	♀	Fibrocystic pancreatitis	−1	+3
7	J. Mc.	8	♂	Poor appetite, vasomotor rhinitis	−2	+2
8	S. S.	6	♀	Behavior problem, poor appetite	−3	+1
9	O. M.	3	♂	Behavior problem, poor appetite, adopted child	−3	+2
10	H. P.	8	♂	Asthma, eczema, hay fever, food allergy, endocrine deficiency	−4	+4
11	M. A.	10	♀	Obesity, vasomotor rhinitis, undersize	+4	0
12	D. C.	9	♂	Anterior poliomyelitis at 6 yr.	−2	+4
13	M. C.	6	♀	Feeding problem (from birth)	−4	+4
14	L. K.	10	♂	Under height for age, behavior problem	Normal	0
15	E. C.	8	♀	Asthma, behavior problem	−3	+1
16	P. N.	9	♀	Twin, congenital heart	−4	+3
17	S. W.	7	♀	Asthma, behavior problem	−3	+2
18	J. F.	6	♀	Asthma	−3	+3
19	G. S.	7	♀	Asthma, hay fever	−4	+4
20	A. H.	7	♂	Asthma, hay fever, behavior problem	−3	+2
21	P. K.	8	♂	Vasomotor rhinitis, behavior problem	+1	+2
22	P. B.	8	♂	Normal. Tall and thin from first year	+4	+2
23	A. W.	4	♀	Feeding problem from birth	−3	+3
24	J. R.	3	♀	Severe asthma	−2	+2
25	N. M.	6	♀	Eczema, hay fever, adopted child	−2	+1
26	C. T.	3	♀	Low milk intake, tuberculosis contact, calcified mediastinal glands, tall	−3	+3
27	B. N.	7	♀	Severe eczema, hay fever, asthma, dietary deficiencies due to food allergy	−3	+3
28	J. C.	8	♀	Behavior problem	−3	+1
29	C. M.	6	♂	Urticaria, G.I. allergy, overactive, excitable	−3	+2
30	P. R.	6	♂	Progressive nerve deafness, vasomotor rhinitis	−3	+3
31	D. M.	3	♀	Asthma, undersize	−3	+2
32	B. S.	7	♂	Asthma	−3	+3
33	P. M.	7	♂	Severe asthma	−4	+4
34	D. S.	7	♂	Vasomotor rhinitis, sialorrhea	−4	+4
35	R. C.	6	♂	Urticaria, G.I. allergy, hay fever, undersize	+2	+1
36	M. P.	6	♀	Urticaria, hayy fever, G.I. allergy	−3	+2
37	D. E.	4	♂	Convulsions, hypothyroidism, calcium deficiency. Adopted child	−4	+4
38	E. G.	11	♂	Vasomotor rhinitis, food allergy, behavior problem	−4	+2
39	R. C.	8	♂	Severe asthma, hay fever	−2	+2
40	M. R.	6	♀	Repeated respiratory infections, undersize for age	−3	+3

CHILDREN ADMITTING TO PRESENT STUDY

CLINICAL RESPONSE +1 TO +4	RELIABILITY +1 TO +4	HEIGHT CHANGES (IN.)			WEIGHT CHANGES (LB.)		
		1 YEAR BEFORE R	1ST YEAR AFTER R	2ND YEAR AFTER R	1 YEAR BEFORE R	1ST YEAR AFTER R	2ND YEAR AFTER R
+3	+4	1	1¾	2¾	4	4½	2
+3	+4	1	1½	2	4	3¾	2
+3	+2	1	3	3	4	12	2
+4	+4	1½	3	3	4	9	7
+4	+3	1	3	5½ (15 mo.)	8	13	33½
+4	+4	1	2½	2	2	7	6
+4	+4	1½	2½	4½	8	10	18
+3	+3	1½	2½	No reply	6	10	No reply
+3	+4	2	4	1¾	3	5½	6
+2	+4	2	1⅝	2	2½	4	2
0	+4	1	2¼	No reply	14	9½	No reply
+3	+4	¼	4	2	2	11½	4
+3	+4	1¼	3	1¾	2	2½	½
+2	+4	2	3	No reply	4½	8	No reply
+3	+4	1½	2	2¾	6	8	12
+4	+4	1	3	1	5	9	1
+4	+4	2	2¼	2	2	5	5
+4	+4	1	2¼	2 (6 mo.)	3	7½	10
+4	+4	1	4	1	3	13	0
+4	+4	1¼	2½ (9 mo.)	3	5	10½	6
+3	+4	1¼	2½	2¾	5	0	8
+3	+4	2½	2½	1½	5	5½	4
+3	+4	2¼	2	3	2½	5	0
+4	+4	1½	3¼	1½	5	3	2
+4	+4	¾ (7 mo.)	2½	1½	0	11½	7
+3	+2	2¼	2¼		5½	6½	
+4	+4	1¾	1¾	2¾	3½	5	12½
+4	+4	2 (10 mo.)	1½ (13 mo.)	2½	7½	7	10
+4	+4	1¾	1¾	3	9½	1	4
+3	+4		2			10	
+4	+3	1 (3 mo.)	1¾	No reply	2	3½	No reply
+4	+4		1½	No report since		5	No report since
+4	+4		2¾			9	
+4	+3	1	3¼	1	1½	13½	1½
+4	+4	1½	2¾	Not long enough	3	5	Not long enough
+4	+4	½	3	2	3	8½	4
+4	+4	4½	3	3	5	8	Lost 3
+4	+4	4	3½	No report since	7½	9½	No report since
+4	+4	2½	3	4	4	11	7
+4	+4		2¼	½ (7 mo.)		5	3

TABLE

CASE NO.	INITIALS	AGE AT R	SEX	DIAGNOSIS	REPORTED APPETITE −4 TO +4	ESTIMATED UNDER NUTRITION 0 TO −4
41	L. R.	7	♀	Asthma, hay fever, G.I. allergy, vasomotor rhinitis	−2	+2
42	M. L.	8	♀	Obesity, short stature	+1	
43	J. G.	3	♂	Eczema, asthma, hay fever	−3	+3
44	R. R.	7	♂	Vasomotor rhinitis, overactive, excitable	−3	+3
45	F. P.	3	♂	Eczema, undersize	−2	+3
46	D. M.	2	♂	Markedly undersize, vomiting (behavior), premature	−4	+4
47	J. R.	3	♂	G.I. allergy	−4	+3
48	J. L.	3	♂	Asthma, eczema, hay fever	−3	+3
49	J. L. H.	8	♂	Severe asthma, hay fever, eczema	−2	+2
50	L. R.	7	♀	Vasomotor rhinitis, G.I. allergy	−3	+2

administration of the supplement. The grid records were first separated into three groups:

Group I. Thirteen children showing progressive loss of physique before B_{12} and B_1 were prescribed This was evidenced by a drift toward the right in the physique channels on the Wetzel Grid.

Group II. Five children who were below normal but "holding their own" before but showed gain of physique after therapy. The grid plots were moving in a straight line within a B channel before treatment and shifted to the left after treatment.

Group III. Fourteen children who did not display any growth failure but were under par physically. Their grid picture remained relatively unchanged after therapy.

Table II summarizes the responsiveness of these three groups as determined by Wetzel Grid analysis. The positive (+) responses are those who showed definite increase of physique (channel shift to left) and increase in auxodrome after B_{12}-B_1 supplementation. The equivocal responses (±) are those who showed slight gains; while the negative (−) responses indicate that no change occurred in the growth rate of the child.

Table III summarizes the change in developmental levels per month before and after therapy for the three groups. Wetzel (1953) makes the point that gaining one developmental level per month is not a sufficient promise for the inference that growth is normal. Constant physique, as shown by upward progress in the same channel, must be maintained simultaneously with n o r m a l growth rate before growth failure can be ruled out.

As previously stated, these children were grouped according to maintenance or loss of physique. It is interesting to note that the average developmental levels of all groups would indicate that their rate of gain was normal before therapy. However, Groups I and II, who were below normal in physique, were the only ones showing increase in developmental rate after therapy. Group III, which could be considered "normal" by both standards (physique and developmental rate), did not react to B_{12}-B_1 therapy.

From the tables, it is seen that the developmental progress before treat-

I—CONT'D

CLINICAL RESPONSE +1 TO +4	RELIABILITY +1 TO +4	HEIGHT CHANGES (IN.) 1 YEAR BEFORE R	1ST YEAR AFTER R	2ND YEAR AFTER R	WEIGHT CHANGES (LB.) 1 YEAR BEFORE R	1ST YEAR AFTER R	2ND YEAR AFTER R
+4	+4		2¼			2½	
		1½	2	Will not report	4	7½	Will not report
+3	+4	1	2½	Will not report	7	3	Will not report
+4	+4	1¾	3¼	2½	5	9	8
+3	+3						
+3	+4	5½? (10 mo.)	2¼	No report since	3¼	3¾	No report since
+4	+4	2	3½	2½	3½	7	9
+3	+4	1	3	4	5	5½	5
+4	+4	4	2½	1¼	2	5½	8½
+4	+4	2	2½	3½	8	8	10

TABLE II. SUMMARY OF RESPONSES TO B_{12}-B_1 SUPPLEMENTATION IN CHILDREN PLOTTED ON WETZEL GRIDS

CASE NO.	AGE	DIAGNOSIS	RESPONSE 1ST YEAR	RESPONSE 2ND YEAR
Group I. Loss of physique and slowed auxodrome				
3	7	G.I. allergy	+	Maintained
5	12	Asthma, urticaria	+	+
7	8	Poor appetite, vasomotor rhinitis	±	+
12	9	Anterior poliomyelitis at 6 yr.	+	Maintained
15	8	Asthma, behavior problem	±	+ (Steady improvement)
16	9	Twin, congenital heart	-	-
20	7	Hay fever, asthma, behavior problem	+ (Slight)	+
25	6	Hay fever, eczema, adopted child	+	+
27	7	Hay fever, asthma, severe eczema, dietary deficiencies due to food allergy	-	+
34	7	Vasomotor rhinitis, sialorrhea	+	Maintained
36	6	Hay fever, urticaria, G.I. allergy	+	+
39	8	Hay fever, severe asthma	±	-
50	7	Vasomotor rhinitis, G.I. allergy	-	+
Group II. B channels with gain of physique on therapy				
17	7	Asthma, behavior problem	-	+ (Slight)
19	7	Hay fever, asthma	+	±
28	8	Behavior problem	+	+
44	7	Vasomotor rhinitis, behavior problem	-	±
47	3	G.I. allergy	+	+
Group III. Under-par children without simple growth failure prior to therapy				
1	6	Twin, congenital heart	-	-
2	6	Twin, congenital heart	-	-
4	8	Feeding problem, behavior problem	±	+ (Not related to B_{12}-B_1)
6	6	Fibrocystic pancreatitis	+ (Acceleration)	
8	6	Poor appetite, behavior problem	±	-
9	3	Poor appetite, behavior problem	±	
10	8	Hay fever, asthma, eczema, food allergy, endocrine deficiency	-	
14	10	Under height for age, behavior problem	-	
18	6	Asthma	-	?
21	8	Vasomotor rhinitis, behavior problem	-	-
23	4	Feeding problem from birth	+(?)	
29	6	Urticaria, G.I. allergy, behavior problem	-	-
30	6	Progressive nerve deafness, vasomotor rhinitis	-	
35	6	Hay fever, urticaria, G.I. allergy, undersize	-	-

ment in Group I (those with the characteristic pattern of malnutrition) was on the average 1.03 levels per month before therapy and 1.33 levels per month after therapy. In Group II (those children who gained physique coincidental with the supplement administration), the respective rates were 1.16 levels per month and 1.53 levels per month. In contrast, the rates in Group III ("nonreactors") were 1.24 levels per month before therapy and 1.04 levels per month during the year of therapy.

Tests of statistical significance for the difference between the means of changes in developmental levels before and after B_{12}-B_1 administration were made. For Group I, $t = 2.30$ and $.02<p>.05$. For Groups I and II combined, $t = 6.64$ and $p<.01$. With Group III, $t = 1.67$ and $.1<p>.2$. Thus the gains registered in Groups I and II are statistically significant, while the average course in Group III was not appreciably altered during the first supplement year.

Table IV shows the average height and weight gains of the three groups before and after therapy. It is evident that the increments of the first two groups far exceed those of Group III.

Fig. 1 compares graphically the developmental rates before and after B_{12}-B_1 supplementation.

TABLE III. DEVELOPMENTAL LEVEL PER MONTH (AVERAGES)

	BEFORE R	AFTER R	DIFF.	T VALUE*
Group I	1.03	1.33	+.33	2.30 (.02<p>.05)
Group II	1.16	1.53	+.37	6.64† p = <.01
Group III	1.24	1.04	-.20	1.67 .1<p>.2

*Fisher's "t" test for comparison of two groups of equal size.
†Average of Groups I + II.

TABLE IV. AVERAGE RATES OF HEIGHT AND WEIGHT GAINS BEFORE AND AFTER R

	HEIGHT RATES			
	INCHES/MO.		INCHES/YR.	
	PRE-R YEAR	R YEAR	PRE-R YEAR	R YEAR
Group I (13 cases)	.23	.28	2.8	3.4
Group II (5 cases)	.20	.26	2.4	3.1
Group III (14 cases)	.19	.20	2.3	2.4
Total (32 cases)	.21	.25	2.5	2.6
	WEIGHT RATES			
	POUNDS/MO.		POUNDS/YR.	
	PRE-R YEAR	R YEAR	PRE-R YEAR	R YEAR
Group I (13 cases)	.42	.79	5.0	9.5
Group II (5 cases)	.47	.69	5.6	8.3
Group III (14 cases)	.44	.39	5.3	4.7
Total (32 cases)	.44	.60	5.3	7.2

B. *Analysis by Tuxford Method.*—To study the results further, a modification of Tuxford's method[6] for estimating the physique and nutrition of children was employed. The exact modifications will form the basis of another report analyzing various indices of growth. This method is based on *positional* changes in contrast to

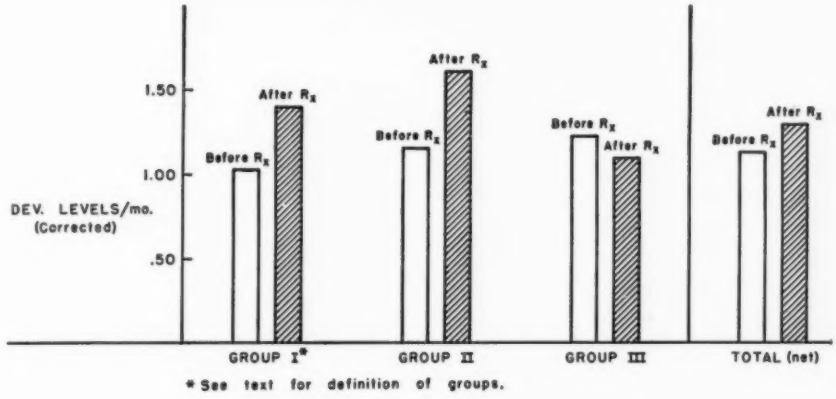

Fig. 1.

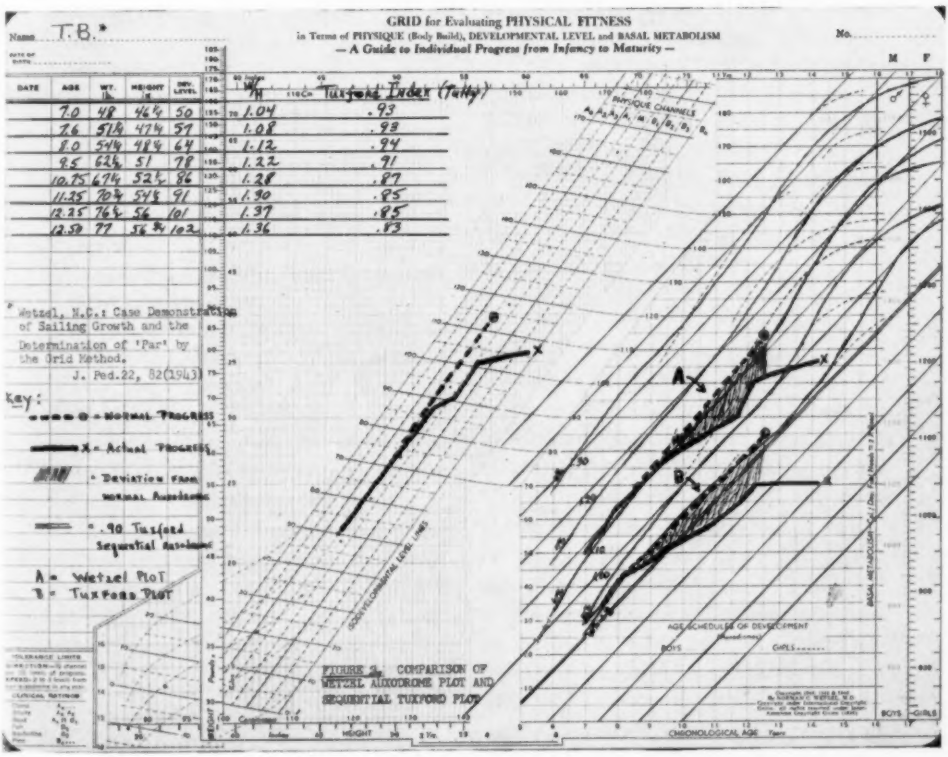
GRID for Evaluating PHYSICAL FITNESS
in Terms of PHYSIQUE (Body Build), DEVELOPMENTAL LEVEL and BASAL METABOLISM
— A Guide to Individual Progress from Infancy to Maturity —
Name T.B.*
No.
DATE AGE WT. HEIGHT DEV. LEVEL
7.0 48 44½ 50
7.6 51½ 47¼ 57
8.0 54½ 48¼ 64
9.5 62½ 51 78
10.25 67¼ 52½ 86
11.25 70¾ 54⅝ 91
12.25 76½ 56 101
12.50 77 56 3/4 102
Tuxford Index (Tully)
1.04 .93
1.08 .98
1.12 .94
1.22 .91
1.28 .87
1.30 .85
1.37 .85
1.36 .83
Wetzel, N.C.: Case Demonstrating of Sailing Growth and the Determination of 'Part' by the Grid Method. J. Ped. 22, 82 (1943)
Key:
= Normal Progress
X = Actual Progress
= Deviation from Normal Expectancy
= .90 Tuxford Sequential Increment
A = Wetzel Plot
B = Tuxford Plot
PHYSIQUE CHANNELS
AGE SCHEDULES OF DEVELOPMENT
BOYS
GIRLS
CHRONOLOGICAL AGE
FIGURE 2. COMPARISON OF WETZEL AUXODROME PLOT AND SEQUENTIAL TUXFORD PLOT.

Fig. 2.—Comparison of Wetzel auxodrome plot and sequential Tuxford plot.

Wetzel's *rate* changes. It deserves mention that the results obtained by grid standards were corroborated by this totally different technique in all but one case (Fig. 2).

DISCUSSION

It is clear from the results obtained in this study that chronically ill children do not differ substantially from those with no evidence of disease in their pattern of response to vitamin B_{12} therapy. In some cases, the arrest of growth failure occurred during the first year with recovery of substance and physique evident the second year. In Group I (those children showing a definite pattern of malnutrition), nine out of twelve showed some degree of response during the first year of supplementation, while eleven out of thirteen were improving or holding their gains in the second year. Out of the whole group of thirty-two, there was definite reversal of trend with B_{12}-B_1 in twelve children; this figure increased to sixteen probable positives by the second year. Thus, with patients classified according to the Wetzel criteria, a significant improvement in malnourished children took place when they were given supplements of 25 μg of B_{12} and 10 mg. of B_1 orally each day.

These results compare favorably with those reported by Wetzel (1953) in which twenty-three of thirty-six underdeveloped children, considered "normal" in that they presented no clinical evidence of disease processes, responded positively to oral B_{12} therapy. In the present study, 50 per cent of the thirty-two children followed by the Wetzel Grid technique evidenced quantitative gains which could be evaluated numerically. By grid analysis, eighteen of these thirty-two children had demonstrated simple growth failure. Sixteen of these eighteen children (88 per cent) responded by positive channel and auxodrome shifts, while the fourteen children whose charts did not reflect growth failure showed no measurable response to the therapy.

All of the children studied gave a clinical impression of improved nutritional status with reports of increased appetite and concomitant weight gains. Analysis of the grid data, however, indicates that an actual increase in growth rate occurred only when growth failure was genuinely present prior to treatment. The greater gains achieved by those in Group I substantiate the observation by other investigators that the degree of response parallels the need for improvement. It has never been contended that B_{12} therapy will cause a normal child to become obese. Rather, the preferential action of the vitamin seems to be directed toward the simple growth failures which are responsive to an increased food intake. Vitamins B_1 and B_{12} have other physiological roles in addition to their mutual appetite-stimulating effect. The maintenance of a normal carbohydrate metabolism and a properly functioning gastrointestinal tract by thiamine and the regulation of protein and lipid synthesis by vitamin B_{12} are important adjuvants to recovery from depressed growth.

SUMMARY

1. Vitamin B_{12} (25 μg) and vitamin B_1 (10 mg.) were administered daily to fifty children with a clinical

diagnosis of malnutrition and anorexia due to chronic illness. By clinical impression, responses in these children seemed to indicate a general improvement in appetite coupled with increased weight and height increments over pretreatment averages.

2. Analysis by Wetzel Grid charts prepared for thirty-two of these patients revealed positive channel and auxodrome shifts in 88 per cent of eighteen children whose charts indicated a simple growth failure, but no appreciable response in fourteen children whose charts did not.

3. The improvement in monthly developmental levels was shown to be statistically significant with a probability of error less than .01, hence, the improvement must be attributed to therapy rather than chance.

4. The inability to discriminate between actual and apparent growth failure by clinical impression emphasizes the need for objective standards in appraising response to nutritional supplements.

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ADDENDUM

Since completion of this study, essentially similar results have been obtained with oral B_{12} supplementation by Larcomb and his associates (J. PEDIAT. **45**: 70, 1954) in a group of institutionalized deaf and blind school children. The correlation between results achieved with a controlled diet and one of free choice further confirm the orexigenic properties of the compound.

RECURRENT BACTERIAL PAROTITIS

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BEFORE the availability of specific serological tests for mumps, many children with recurrent swelling of the parotid gland were thought to be suffering from second or third attacks of mumps. It is now clear that in most instances some other etiology must be found, and that second attacks of mumps which can be confirmed in the laboratory are still to be reported.[1] It is usually easy to exclude salivary calculi, neoplasms, and leukemia as causes for the recurrent swelling. However, it is not so easy to evaluate the place of bacterial infection in this syndrome. In 1940 Payne[2] discussed recurrent parotitis and felt that it was due either to a *Streptococcous viridans* or a pneumococcus. In his experience, the more severe cases were usually a result of pneumococcal infection. More recently, Bigler,[3] Jones,[4] and Royce[5] reported on the syndrome of recurrent parotitis in children. *Str. viridans* was recovered from the parotid secretions of each of Bigler's 100 cases.[3] Jones[4] found this same organism in fifteen of seventeen patients, but pneumococci were also present in three, and were the only bacteria in the two other patients. There was a less uniform bacterial flora in the twenty-two children studied by Royce.[5] Despite the consistent finding of bacteria in the parotid secretions of these patients, therapy with antibacterial agents has been notoriously disappointing. Consequently, recurrent parotitis remains an intriguing and sometimes very annoying problem.

It is the purpose of the present communication to consider this syndrome of recurrent bacterial parotitis. To illustrate different aspects of the disease, the case histories of three children will be reported. To aid in an understanding of the refractory nature of these infections, the normal anatomy and physiology of the parotid gland will be reviewed and the results of sialograms and a biopsy from one of the patients will be presented. Because each of these three children either gave a history or had a positive complement fixation test for mumps, the role of a preceding mumps infection in the pathogenesis of certain instances of recurrent parotitis will be discussed. Finally, suggestions will be made for the clinical management of this disease.

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CASE REPORTS

CASE 1 (An example of abscess formation).—J. Z. (a patient of Dr. E. Arnheim) had mumps at the age of 2 years. He recovered spontaneously but had some residual painless swelling in the right parotid area which enlarged and regressed from time to time. However, two weeks prior to

admission at age of 5½ years, he was struck by a fist in this area and shortly afterward developed a marked swelling and pain. On admission to the Mount Sinai Hospital, his temperature was 100° F. The right side of the face was markedly swollen. A smooth, firm, well-demarcated warm mass was described as the "size of an egg." The center was fluctuant. No inflammation was seen at the opening of Stensen's duct. The white blood cell count was 7,650 per cubic millimeter with 68 per cent segmented forms. Incision and drainage were performed by Dr. Arnheim and approximately 50 c.c. of thick greenish pus was obtained which on culture contained only *Neisseria catarrhalis*.

Five months later the child was readmitted because of a return of the swelling. This time, some thick gelatinous fluid was aspirated from which no bacteria were grown.

Case 2. (An example of diffuse suppurative parotitis).—E. McD. gave no definite history of having had mumps. However, she had had her first attack of bilateral parotid swelling at the age of 4 years. This was said to have been associated with a purulent drainage into the mouth. She responded to treatment with intramuscular penicillin. Four months later she had a second attack of parotitis, this time limited to the left side. She again responded to penicillin. Fourteen months later she had a third episode, this time involving only the right side. Her fourth episode occurred at the age of 5½ years, when she was admitted to the Mount Sinai Hospital. On admission, her temperature was 102° F., and there was a marked swelling of the left parotid. The secretion from this gland was purulent, and on culture *Staphlococcus albus* and *Esch. coli* were recovered. The white blood cell count was 13,353 per cubic millimeter with 83 per cent segmented forms. The sedimentation rate was 28 mm. in one hour. The infection was arrested by treatment with penicillin given intramuscularly and Aureomycin orally.

Although, as noted, there was no definite history of mumps, the child's complement fixation test for mumps was positive in titer of 1:16. This was interpreted to mean that one of her earlier attacks of parotitis was most likely mumps. Her sialogram showed a pattern of fine stippling indicative of inflammatory disease (similar to that shown by patient M. V. in Fig. 1). At the time of discharge she was asymptomatic and her white blood count was 5,150 per cubic millimeter with 32 per cent segmented forms.

Case 3. (An example of indolent parotitis).—M. V. was seen for the first time in March, 1950, when he was 6 years 10 months of age. There was a history of his having had mumps with bilateral parotitis approximately two years earlier. During the year which followed there were several minor episodes of swelling, especially in the right parotid region, ascribed to the eruption of a sixth year molar. He was then free of any swelling for nine months until Dec. 15, 1949, when he was again thought to have mumps with bilateral parotitis. Approximately one month later there was an episode of swelling in the region of the right parotid gland. The child's temperature was 100° F. He was treated with ten daily injections of penicillin and given a sulfonamide preparation. The swelling subsided, only to recur about five weeks later. This time he was treated with Aureomycin for six days. The swelling again subsided but recurred bilaterally about one month later. He was admitted to another hospital and again treated with Aureomycin. The swelling once more subsided in a few days and he was then referred to the Mount Sinai Hospital for further study. At that time the physical examination was essentially negative except for a mild rhinitis and a postnasal drip. The openings of Stensen's ducts were not inflamed. A watery secretion was obtained from the ducts which did not contain any pus cells or eosinophils. Blood was drawn for

a mumps complement fixation test. A titer of 1:128 was obtained, which confirmed the child's story of having had mumps, although, of course, one could not ascertain whether it was the illness in 1948 or 1949. A sialogram was made (Fig. 1) by injecting 0.5 c.c. of Lipiodol into the right duct orifice. The main duct was found to be normal in caliber, shape, and outline. The arborization of the secondary radicles was also normal. However, throughout the substance of the gland there were tiny droplets of Lipiodol, indicating cystic dilatation of the ductules. This droplet forma-

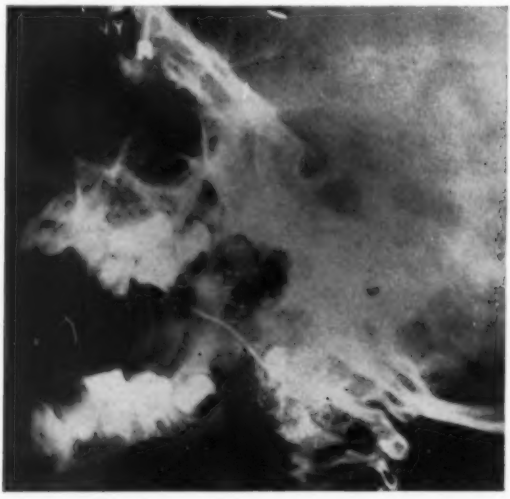

Fig. 1.—Sialogram of parotid of patient M. V. (Case 3) showing abnormalities in distal duct system. The droplets of Lipiodol are characteristic of cystic dilatation of ductules.

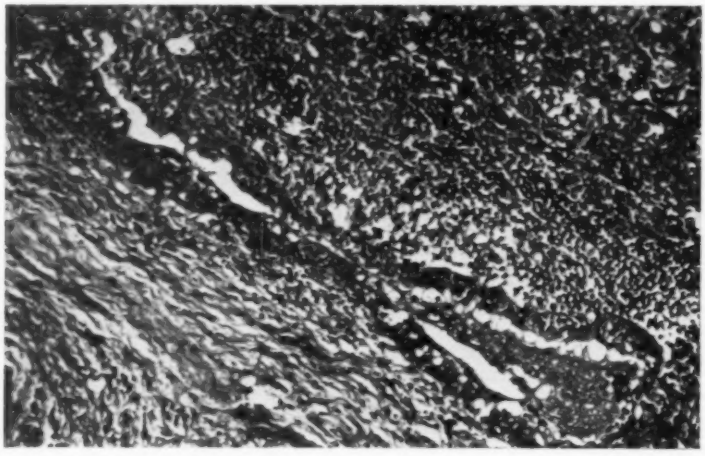

Fig. 2.—Photomicrograph of tissue from parotid of patient M. V. (Case 3) showing changes in epithelial cells of duct and adjacent infection.

tion was interpreted as being characteristic of inflammation of the parotid gland. There was no x-ray evidence of a calculus. Because there was no obvious swelling at the time, the child was sent home with the suggestion that if he suffered another episode, he be admitted for further investigation. He was admitted to the Mount Sinai Hospital in October, 1950, on the third day after the onset of swelling of the right parotid. At that time he was afebrile although there was a soft, tender swelling over was obtained of the gland and a portion was also inoculated into embryonated chicken eggs without recovery of mumps virus. The biopsy specimen was described as showing evidence of acute and chronic nonspecific inflammation with slight dilation of the ducts. The changes in the duct epithelium and the periductal inflammatory reaction are clearly shown in Fig. 2. As will be discussed later, the over-all histologic picture was not unlike that described by Weller and Craig[12] after uncomplicated mumps.

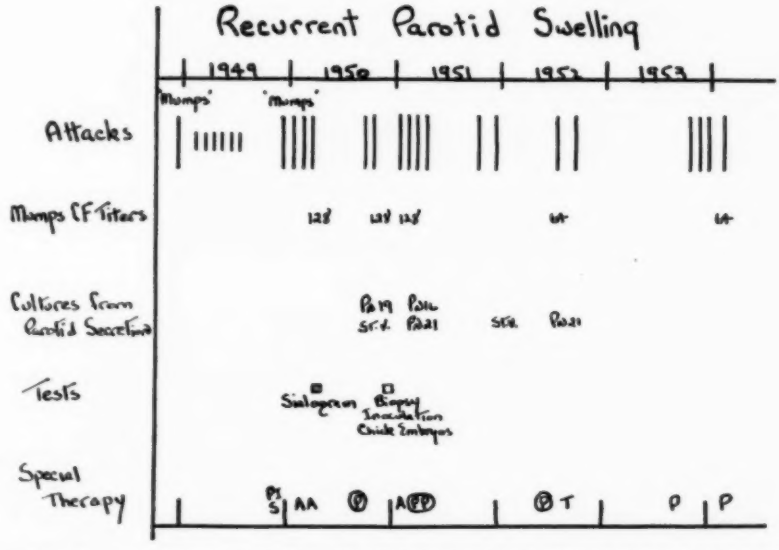

Fig. 3.—Graphic representation of clinical course, significant laboratory studies, and special therapy of patient M. V. (Case 3). *Pn*, pneumococcus; *St. v.*, *Streptococcus viridans*; *A*, Aureomycin; *PI*, penicillin intramuscularly; *P*, penicillin orally; Ⓟ, penicillin instilled into ducts; *S*, sulfonamide; *T*, Terramycin.

the angle of the right jaw and slightly below it. The white blood cell count and differential were normal. The sedimentation rate was 4 mm. Total protein was 6.3 (albumin 3.7, globulin 2.6). The culture of secretion from the parotid duct showed pneumococcus type 19 and *Str. viridans*. These same organisms were recovered from the child's throat. Parotid fluid was collected and inoculated into chick embryos, but no mumps virus was recovered. Subsequently, a biopsy

In view of the finding of pneumococci in the parotid section and because of the superficial similarity of the sialogram to bronchiectasis, direct instillation of crystalline penicillin into Stensen's duct was attempted. This was done twice in the hospital and three times at home after discharge. The child's subsequent course is best illustrated in the chart (Fig. 3). He has continued to have parotid swellings. The parotid secretions have been cultured on several

occasions and a variety of bacteria discovered. Similarly, a variety of antibiotics have been employed without noticeably influencing the course. A typical episode was that which started on Feb. 22, 1954, and which probably represented his nineteenth attack. The day before he had been out in the rain. The next day he said his mouth felt full on the inside and he thought he was going to have another attack. By evening there was a moderate but definite swelling on the right side. He complained of pain in this region when he talked or moved his mouth. The next day the other side was also moderately swollen. For two days he had trouble eating. His temperature never went above 100° F. and he did not appear very sick. After three days the swelling disappeared. During the acute phase the child took Bicillin orally. Although attacks usually follow respiratory infections, not all upper respiratory infections have been followed by parotid swelling.

DISCUSSION

The cases just presented show that recurrent bacterial parotitis may take at least three different forms or degrees of intensity, namely, abscess formation, diffuse suppuration, and indolence.

The systemic reaction of the patients may vary from mild discomfort to severe pain, from low-grade fever to temperatures in the range of 102° F., from normal leukocyte counts to leukocytosis with a shift to the left. The parotid secretion may or may not contain frank pus, and a variety of microorganisms may be recovered. The constant features are recurrent parotid swellings without evidence of obstruction by a calculus, no other systemic disease, and destruction of parotid epithelium as reflected in abnormal sialograms or abscess formation.

There are several aspects of this syndrome which require further consideration. These include why it occurs so relatively infrequently and why, when it does occur, it is so difficult to eradicate. The answer to these questions seems to lie in an understanding of the unique anatomical structure and function of the parotid gland.

The parotid gland is the largest of the salivary glands. It arises by the ingrowth of oral epithelium into the underlying mesenchyme. The start of this ingrowth is at about the sixth week of embryonic life. When it reaches the area of the ramus of the mandible, the main duct begins to branch freely into the primordial cell cords which form the branch ducts and the terminal alveoli. The epithelium of the terminal buds forms the secreting glandular epithelium, while that of the stalk forms the lining epithelium of the duct. These buds are at first solid and do not become hollow until about the twenty-second week of intrauterine life.[6] In the infant and child, the gland lies on the side of the face, below and in front of the ear, the mastoid process and sternocleidomastoid muscle. It forms a lobulated mass with a large triangular superficial surface. The parotid duct runs across the masseter muscle. At the anterior border of the masseteric muscle, it turns abruptly and runs inward through the fat of the cheek to the buccinator muscle which it pierces. It then passes for 5 to 10 mm. between the buccinator muscle and the mucous membrane, and opens into the vestibule of the mouth by a very small orifice, on a variously developed papilla, opposite

the second upper molar tooth. The duct itself measures from 38 to 62 mm. in length and 3 to 4 mm. in diameter. Its caliber is very much wider than its orifice.[7] This fact and its long course are significant anatomical barriers to ascending infection. The normal duct system is perhaps best demonstrated by the sialogram shown in Fig. 4. The basic structure of the gland is the cluster of secretory cells, sometimes called endpieces, around collecting ductules. These ductules differ from those of other salivary glands in having areas of marked narrowing ("intercalatid ducts"),[8] which may account for the persistence of infection once it reaches the secretory cells. The normal histology is shown in Fig. 5.

Fig. 4.—Sialogram of normal parotid gland showing duct system.

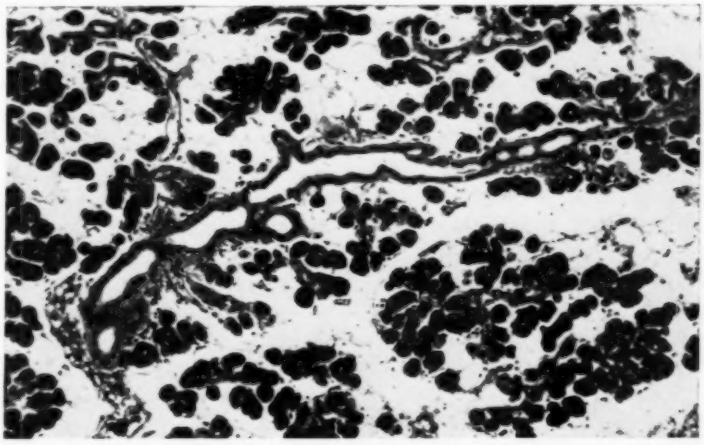

Fig. 5.—Photomicrograph showing ducts and glandular tissue in normal parotid gland.

The function of the parotid is to secrete a fluid which furnishes protection, moistening, and cleansing of the intraoral structures, and enzymes which aid in the digestion of certain carbohydrates and nucleoproteins. The parotid saliva is a clear, thin liquid. It is normally composed of water, salts, enzymes, and very little protein. It does not contain mucin—which sharply differentiates it from the secretion of the other salivary glands, and points up the fact that the parotid contains only serous secreting cells while other salivary glands have both mucous and serous secreting cells. Mucin is believed to have some antibacterial activity and its absence in parotid secretion, just as the narrow collecting ductules, probably serves to favor persistence of bacterial infection once it is established. The pH of the secretion of the normal resting gland is 6 to 8; and the concentration of inorganic salts varies from 30 to 80 per cent of their circulating blood values. The fluid is normally sterile.[9]

It is said to be very difficult to infect the normal gland by the retrograde injection of bacteria by way of Stensen's duct, unless the duct is blocked immediately after injecting the organisms.[10] The inherent ability of the gland to resist ascending bacterial infection is believed to be due to the continual secretion of saliva which washes organisms away, as well as to the length and shape of Stensen's duct and its narrow orifice which also normally hinder the retrograde flow of mouth fluids.

When bacterial infections of the parotid do occur, it is logical to look for some breakdown in the natural defenses. Indeed it is well recognized that bacterial parotitis is more likely to occur in newborn infants who are dehydrated and in whom the salivary flow is reduced. Because each of our three patients gave some evidence of having had mumps, we were led to inquire what effect this viral infection might have on the natural resistance of the parotid gland to bacterial infection.

There are relatively few reports of the effect of the virus of mumps on the human parotid, since patients rarely die during the acute phase of the disease. However, Weller and Craig[12] recently described the gland of a 9-year-old girl who died of another cause approximately seventy hours after the appearance of soreness in the parotid area and eighteen hours after parotid swelling was observed. Since the mumps virus was isolated from this gland, their description is worth noting in detail. "The gland was not grossly edematous and no petechiae were seen. The general architecture was well preserved. The major alterations in structure occurred in and about the ducts which showed widespread involvement. In the least affected ducts there was a slight swelling of the epithelial lining cells with an increased granularity of their cytoplasm. The lumina contained a small amount of debris consisting of nuclear fragments, polymorphonuclear leukocytes, and desquamated epithelial cells. In the more severely involved ducts, a large portion of the ductal epithelium had desquamated, leaving a bare basement membrane. In the most severely involved region, there was great dilatation of ducts; all of the epithelial cells were desquamated and a large mass of nuclear and cellular debris

choked the lumen. The inflammatory reaction spread for a short distance out into the periductal connective tissues and tangentially involved the adjacent acini. The interstitial exudate contained little or no fibrin, yet the periductal edema was moderate to marked in degree. In the periductal regions, the lymphocyte was the predominant inflammatory cell; a few large mononuclear and rare polymorphonuclear cells are present. The difference in the inflammatory cell types without and within these ducts was striking." In the main, these are the findings which had been reported by earlier pathologists.[11] These pathologic changes, especially the degeneration of the ductal epithelium, might be expected to predispose to a secondary bacterial infection.

Although bacterial parotitis has been said to be a rare complication of mumps,[11] in the absence of any other obvious explanation for the onset of the disease in our patients, it seemed worth exploring the possibility that mumps might have been the predisposing factor.

It will be recalled that two of our three patients had positive serological tests for mumps. Since these children were not seen by us and no serological tests were done until approximately two years after the first attack, it was not possible to determine which attack of parotitis was mumps. Each recognized attack of parotitis in patient 2 (E. McD) was said to have been associated with a purulent discharge. She had a low titer of antibody, and so it is possible that her difficulty was ushered in by a preceding mild and unrecognized episode of mumps parotitis. Many of the attacks of parotid swelling experienced by patient 3 (M. V.) were seen by us, and several could easily have been mistaken for mild attacks of mumps. Evidence that this did not represent recurrent mumps lies in our inability to demonstrate a rise in mumps complement fixing antibody after these attacks, such as is seen in recurrent herpes simplex, and in our failure to recover the virus from the parotid fluid and tissue. Although we cannot prove that any of these three instances of recurrent bacterial parotitis followed an initial mumps parotitis, the possibility remains inviting. Because the pathology reported above suggests that mumps might predispose to bacterial parotitis, and the ready availability of serological tests for mumps, it should now be of interest to investigate every child experiencing a second attack of parotitis with this in mind. In this way the role of mumps in predisposing to this disease might actually be defined.

The role of bacteria in recurrent bacterial parotitis seems to be that of a secondary invader, once the natural defenses of the parotid have been overcome. As in patient 3 (Fig. 3), the organisms recovered may vary from time to time, and may reflect the flora of the mouth.

The treatment of recurrent bacterial parotitis depends upon the type of infection. As in the cases presented, occasionally surgical drainage, systemic chemotherapy, or no specific therapy is required. The problem is to eradicate residual infection and to prevent recurrences. Because of the superficial resemblance of the sialogram to bronchiectasis, direct retrograde instillation of penicillin has suggested itself to a number of workers. We, too, were

prompted to try this in our third patient without any long-lasting improvement. However, Jones[4] reported more encouraging results after injecting much larger doses than we used. He gave 1 ml. containing 100,000 units of crystalline penicillin daily for six days. Even with his treatment, improvement was limited generally to a reduction in the duration of the periods of activity. Other workers[3] resorted to x-ray treatment of the gland and some, such as Bailey,[13] have even suggested removal of all or part of the parotid when the attacks have been especially troublesome.

From our experience and review of the literature, it would seem that at the present time the most logical treatment consists of the systemic use of antibiotics, and the full utilization of what appears to be Nature's chief defense against bacterial infection, the stimulation of the flow of parotid secretions.

The prognosis for these children is variable. How and why the vast majority eventually get well is not yet clear. It is of interest that two of the mothers of the seventeen children with recurrent bacterial parotitis, reported by Jones,[4] had had the disease in childhood.

SUMMARY

1. The case histories of three children with recurrent bacterial parotitis are presented. Each patient illustrates a different aspect of the syndrome. One had abscess formation, the second, diffuse suppuration, and the third, a relatively mild but indolent infection which, to date, has been responsible for at least nineteen attacks of parotid swelling.

2. The anatomy and physiology of the normal parotid gland are reviewed. The histologic changes which occur following uncomplicated mumps are described. The manner in which these factors predispose to recurrent bacterial parotitis is discussed.

3. Based on what is known of the normal and infected parotid gland, suggestions are made for the clinical management of these patients.

I wish to acknowldege with thanks the assistance of Dr. Norman Simon of the Mount Sinai Hospital who prepared the sialograms; of Dr. Fred Zak of The North Shore Hospital who furnished the slide containing normal parotid tissue; and of Ruth K. Florman who is responsible for the chart presented as Fig. 3.

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NUCLEASES IN THE CEREBROSPINAL FLUID

III. Simultaneous Determination of Ribo- and Desoxyribonuclease in the CSF of Patients with Poliomyelitis

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IN A previous publication it was noted that ribonuclease (RNA-se) was present almost invariably in the CSF of poliomyelitis cases.[1] A lack of desoxyribonuclease (DNA-se) was shown to be a constant feature of similar specimens,[4] among other characteristics.[2, 3] The present report deals with results of simultaneous assays by rapid methods for the two nucleases. Our purpose in this work was to investigate the role of enzymes in the mechanism of neuronal changes during human poliomyelitis, in the hope that a useful diagnostic tool might be developed. Specimens from patients with other conditions were also examined by these techniques and a comparison of these results with those obtained in poliomyelitis reveals a characteristic nuclease pattern in the latter.

METHODS

The spectrophotometric technique of Kunitz[5] for RNA-se was adapted to our purpose. When more than 4 ml. CSF was available, the original direct method was suitable; the routine, however, was different, as described later.

Immediately after mixing, optical density (D) readings were taken at 3,000 Å on a system formed of 0.3 ml. CSF, 2 ml. 0.1 per cent pentosenucleic acid (PNA) in borate buffer pH 7.6,[6] and 2 ml. sterile distilled water. Measurements were taken on a duplicate mixture after thirty minutes' incubation at 60° C. Substrate and CSF controls similarly diluted with buffer were run in parallel in each set of experiments. All tubes were read against borate buffer pH 7.6 and the mean of three consecutive determinations served for the calculations. The D value of the incubated system and controls was deducted from the zero readings. The difference (Δ) was due to RNA-se activity.[5] When the small volume of CSF did not allow the preparation of double assays, the set used for first readings could be incubated in the absorption cells and readings repeated after incubation. Alternative methods for determination of enzyme content are available and total acid soluble phosphorus (TASP) determinations were made as a check on the spectrophotometric method.[1]

The optical determination of DNA-se[7] was not suitable because of autolytic changes in the D of the CSF's[3] which have been largest in the region of 2,600-2,650 Å, where the measurements should be carried out.[10] Therefore, the viscosimetric method[8] was used as previously.[4] Drop in the flow time of the system was recorded, as a measure of DNA-se activity. The maximum experimental error was ± .007, average ± .0025 in the spectrophotometric work and ± 2 seconds in viscosimetry. The PNA was purified from a commercial brand.[9] A highly polymerized preparation* was also assayed with success. The standard enzyme was a commercially available

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*Courtesy G. C. Butler, Department of Biochemistry, University of Toronto.

TABLE I. RNA-SE AND DNA-SE IN CSF SPECIMENS (ILLUSTRATIVE EXAMPLES)

		RNA-se MEASURED BY DECREASE OF OPTICAL DENSITY IN Δ	DNA-se MEASURED BY DECREASE OF FLOW TIME (V) IN SECONDS
Group A: Normal and Neurologically Normal			
1. Normal	Neg. CSF findings	0	0
2. Neurologically Normal	Neg. CSF findings	–0.001	0
3. Pyrexia, origin unknown	Neg. CSF findings	–0.010	3.4
4. Bronchitis, flu	Neg. CSF findings	–0.005	10
5. Gastroenteritis	Neg. CSF findings, Dehydration	–0.013	0
Group B: Meningitis			
6. *H. influenzae* meningitis	Acute, 3,600 cells, polys. Pandy +, growth +	–0.019	35
7. Pneumococcus meningitis	Chronic, 25 cells, lys. Pandy +, growth +	–0.007	0
8. Meningococcus meningitis	Acute, 1,600 cells, polys. Pandy +, growth +	–0.047	10
9. *E. coli* meningitis	Subacute, 800 cells, polys. Pandy +, growth +	–0.032	120
10. Cured tubercular meningitis	Chronic, 2 cells, lys. Pandy trace, growth neg.	–0.011	19
Group C: Poliomyelitis			
11. Nonparalytic polio	Onset 1 wk. before admission, 180 cells, Pandy +, growth neg.	–0.006	0
12. Nonparalytic polio	Acute, 415 cells, 85%, lys. Pandy +, growth neg.	–0.029	0
13. Nonparalytic polio	Onset 4 da. before admission, 300 cells, 60% polys. Pandy +, growth neg.	–0.019	0
14. Nonparalytic polio	Acute, 480 cells, 95% polys. Pandy +, growth neg.	–0.015	0
15. Nonparalytic polio	21 cells, 6 polys. Pandy +, growth neg.	–0.021	0
16. Paralytic polio	Acute, 88 cells, 26 polys. Pandy +, growth neg.	–0.014	0
17. Paralytic polio	Onset 24 hr. before admission, 536 cells, 95% lys. Pandy +	–0.028	0
18. Paralytic polio	Onset 3 da. before admission, 120 cells, 50% polys. Pandy +	–0.030	0
19. Paralytic polio	Acute, 51 cells, 35% lys., weakness developed after admission	–0.013	0
20. Paralytic polio	Subacute, 10 cells, lys. Pandy +	–0.011	0
21. Bulbar polio	Onset 2 da. before admission, 18 cells, Pandy +	–0.034	0
22. Bulbar polio	Onset 1 da. before admission, 99 cells, Pandy neg., growth neg.	–0.035	0
23. Bulbar polio	Subacute, 110 cells, majority lys. Pandy +, growth neg.	–0.027	0
24. Bulbospinal polio	Onset 2 da. before admission, 60 cells, 50% polys. Pandy neg., growth neg.	–0.019	0
25. Bulbospinal polio	Subacute, 45 cells, lys. Pandy +, growth neg.	–0.059	0

TABLE I—CONT'D

Group D: Virus Diseases Other Than Poliomyelitis			
26. Lycytic meningitis	366 cells, lys. Pandy +, growth neg.	−0.032	0
27. Lycytic meningitis	260 cells, lys. Pandy +, growth neg.	0	0
28. Mumps meningitis	400 cells, lys. Pandy +, growth neg.	−0.024	0
29. Mumps encephalitis	60 cells, lys. Pandy +, growth neg.	−0.012	0
30. Guillain-Barré syndrome	No cells, Pandy 4+ (220 mg. %), growth neg.	−0.005	0
Group E: Miscellaneous Neurological and Psychotic Conditions			
31. Huntington's chorea	Neg. CSF findings	−0.023	10
32. Febrile convulsions	Neg. CSF findings	−0.012	0
33. Epilepsy	Neg. CSF findings	−0.033	30
34. Hydrocephalus	2 lys., protein 80 mg. %	−0.029	0
35. Paranoia	Neg. CSF findings	−0.028	41

crystalline RNA-se.† Purified DNA-se and highly polymerized DNA were kindly supplied by Professor Butler.

CSF's were stored frozen, and centrifuged after thawing at 4° C. The neurological group contained both fresh and frozen samples, derived mostly from adults. The remainder were mostly from infants and children.

RESULTS

Simultaneous examination of RNA-se and DNA-se was carried out in 180 individual CSF's. The diagnosis and laboratory findings of the hospital staffs served as the basis for classification into five clinical groups: (a) normal and neurologically normal without any signs of neurological disease or any change in the CSF by routine laboratory procedures; (b) meningitides; (c) poliomyelitis, represented by various clinical forms; (d) virus infections other than polio; (e) miscellaneous neurological and psychotic conditions. Table I presents five examples from each group and subgroup. Low, high, and medium values were chosen to illustrate the trend within the groups. The clinical notes describe the characteristics of the CSF's. Of five normal samples tested for both nucleases, all were negative; in those of neurologically normal persons, both enzymes were present in some, one in others, and none in the remainder. When present, their activity was low (Table I, group A).

The meningitis samples exhibited positive CSF findings, including bacterial growth in the great majority. Both enzymes were present in most instances, the values being higher in the acute stage and lower in the chronic (Table I, group B). The chief clinical form of poliomyelitis is represented by an equal number of samples. The stage of disease varied from one day to three weeks. Great variation was found in cell count, but only one specimen showed negative Pandy. Bacterial growth was absent. The enzyme pattern was characteristic: intense RNA-se and no DNA-se activity (Table I, group C). The examples from group D are selected from CSF's of 8 lymphocytic choriomeningitis, 6 mumps meningitis, 3 mumps encephalitis, 1 measles encephalitis, and 2 Guillain-Barré syndrome cases, one of the latter being blood tinged. The findings were similar to those in polio,

†Supplied by Nutritional Biochemicals Corporation, Cleveland, Ohio.

but RNA-se was at a lower level and sometimes absent. The small numbers, however, warrant no conclusions. The CSF of miscellaneous neurological and psychotic patients displayed high enzyme activity with the frequent presence of both nucleases, without cell increase, and very rarely with some abnormal protein concentration (Table I, group E). The hydrocephalus sample shown is not a typical example, because in other similar specimens DNA-se was also demonstrated.[4] A larger series of samples of neurological conditions will be reported separately.[10]

Table II illustrates the quantitative relationships. The average of RNA-se activity in the normal and neurologically normal group was within the limits of experimental error. There were two exceptions, Δ −.010 and −.013, respectively, which are about twice as great as the mean. The average in paralytic and bulbar polio was about five times higher than in the first group, followed in order by meningitides, miscellaneous neurological, nonparalytic, and other virus samples. There was a definite difference between the various clinical forms of polio, the nonparalytic exhibiting the lowest individual values (in two specimens), the lowest mean and the widest variation in range. The average in CSF's of paralytic and bulbar cases was definitely higher and the standard error lower than in the meningitis group. The coefficient of variation (31 per cent) was the lowest in the paralytic subgroup, which is significant.

The DNA-se findings revealed even more uniform behavior, namely, no detectable enzyme levels at all in virus disease samples (Table II, groups 3 and 4). The average in the normal group is slightly higher than the experimental error. Meningitis specimens showed the widest range, with the highest average, but the standard error of the mean suggested large dif-

TABLE II. ANALYSIS OF RESULTS

GROUP	NUMBER OF CSF	RNA-se ACTIVITY				DNA-se ACTIVITY			
		RANGE IN Δ	AVERAGE ±		STANDARD ERROR	RANGE IN SECONDS	AVERAGE ±		STANDARD ERROR
1. Normal	20	0 to −.013	−.005	±	.001	0 to 10	2.3	±	0.75
2. Meningitis	40	0 to −.062	−.023	±	.002	0 to 120	12.0	±	3.29
3. Poliomyelitis	60								
a. Nonparalytic	20	−.005 to −.042	−.020	±	.002	0	0		0
b. Paralytic	20	−.013 to −.044	−.026	±	.0018	0	0		0
c. Bulbar, bulbospinal	20	−.019 to −.059	−.024	±	.002	0	0		0
4. Virus (other than poliomyelitis)	20	0 to −.032	−.015	±	.0018	0	0		0
5. Miscellaneous neurological and psychotic	20	0 to −.041	−.022	±	.0017	0 to 41	7.2	±	1.09

ferences within the values. Of the forty random samples, twenty-five (63 per cent) displayed some activity. In selected fresh CSF's of acute meningitis cases, the activity was of a higher order (90 per cent).[4] The miscellaneous neurological group was more homogeneous with respect to occurrence and activity of both nucleases. Statistical analysis revealed the results to be highly significant (up to P.01).

Most of the assays were carried out at pH 7.6, which is the optimum range for activity.[11] Many specimens were assayed at pH 5 also. Generally, the former gave better results but, as an exception, higher values were encountered in some nonparalytic polio specimens, at this higher acidity, followed in order by paralytic, and by bulbar polio and meningitis samples (Table III). This is an interesting finding, which has to be verified on a larger number of CSF's. The thermostability of RNA-se and its elevated temperature optimum made a short incubation possible, excluding the danger of contamination.

Table IV presents practical examples from our protocol to underline the reality of the numerical value of the findings, which could be magnified by increasing the CSF volume, time, and temperature of incubation.

Parallelism was observed between the results obtained by the spectrophotometric and the TASP techniques, although the percentage of change with the latter tended to be relatively greater in some instances than with the former (Table V). About 25 per cent of the total phosphorus became acid-soluble, under the influence of active specimens, in thirty minutes. The effect of some CSF's is comparable to that of pure enzymes.[4] Our high values could be simulated by changes brought about by 33 μg of crystalline

TABLE III. RNA-SE IN THE CSF, ASSAYED AT pH 5

GROUP	NUMBER	RANGE OF DECREASE OF DENSITY (Δ)	AVERAGE (Δ)
1. Poliomyelitis			
a. Nonparalytic	5	−.018 to −.051	−.041
b. Paralytic	5	−.007 to −.031	−.022
c. Bulbar, bulbospinal	7	−.019 to −.030	−.025
2. Meningitides	6	0 to −.029	−.015

TABLE IV. EFFECT OF CHANGES IN CONDITIONS OF ASSAYS (ILLUSTRATIVE EXAMPLES)

GROUP	CSF VOLUME (ML.)	TEMPERATURE	TIME	D READINGS (Δ)
1. Nonparalytic polio	0.3	37° C.	18 hr.	−.040
2a. Nonparalytic polio	0.2	60° C.	30 min.	−.015
2b. Nonparalytic polio	1.0	60° C.	30 min.	−.035
3. Nonparalytic polio	1.0	45° C.	4 hr. 30 min.	−.059
4a. Paralytic polio	0.3	30° C.	3 hr.	−.025
4b. Paralytic polio	0.3	45° C.	3 hr.	−.037
5. Bulbar polio	0.4	37° C.	3 hr.	−.046
6. Suspect meningitis	0.3	65° C.	30 min.	−.068
7a. Suspect brain tumor	0.2	65° C.	30 min.	−.011
7b. Suspect brain tumor	0.5	65° C.	30 min.	−.016
8. Suspect herniated disc	0.4	45° C.	30 min.	No change

RNA-se, which caused a decrease of D of –.056 in eight minutes, at instrument temperature.

Correlation of nuclease activity with cell count was observed in the meningitis specimens, but not in the poliomyelitis and the miscellaneous neurological groups (Table VI). The average of cells in the bulbospinal form, for example, was lower than in the nonparalytic, or in meningitis subgroups, but the RNA-se activity was with increase and others with decrease of ultraviolet light absorption. Polio belongs to the first category, meningitis to the second, and the neurological group to the third. This question is under further investigation.[10]

DISCUSSION

The most uniform increase of RNA-se activity was found in poliomyelitis, especially in the paralytic and bulbospinal forms. This is in agreement

TABLE V. RNA-SE MEASURED SIMULTANEOUSLY BY SPECTROPHOTOMETRIC AND BY TASP METHODS

GROUP	NUMBER	DECREASE OF D (Δ)	AVERAGE (Δ)	INCREASE OF TASP MG./ML.	AVERAGE MG./ML.
1. Meningitis	1	–.050	--	.25	--
2. Meningitis	1	–.026	--	.16	--
3. Meningitis	1	–.062	--	.12	--
Total: 16			–.024		.08
1. Nonparalytic polio	1	–.020	--	.10	--
Total: 10			–.020		
2. Paralytic polio	1	–.030	--	.23	.07
Total: 10			–.027		--
3. Bulbar polio	1	–.046	--	.20	.08
Total: 10			–.026		.09

TABLE VI. CORRELATION BETWEEN CELL COUNTS, PROTEIN CONCENTRATIONS, AND NUCLEASE ACTIVITIES

GROUP	TOTAL	CELL COUNT AVERAGE	PROTEIN AVERAGE (MG. %)	RNA-SE AVERAGE (Δ)	DNA-SE AVERAGE (SECONDS)
1. Meningitis	40	500	102.0	–.023	12.0
2. Nonparalytic polio	20	140	58.0	–.020	0.0
3. Paralytic polio	20	170	68.0	–.026	0.0
4. Bulbar polio	20	74	67.2	–.024	0.0

higher in the first. Proteins were increased in all, as a rule. The enzymatically most active CSF's exhibiting only slight pleocytosis came from subacute cases with well-established symptoms, such as weakness or paralysis (Table I, group C).

Finally, a difference was observed in behavior of CSF controls after incubation with regard to change in D.[3] Three types can be distinguished thus far: CSF's with no change, others with our previous findings on a large number of CSF's from polio patients.[1] Relationship with the clinical form and stage is more definite than with the cell count. The ratio of the two nucleases in meningitis samples indicates that these enzymes may be liberated by cell destruction (mostly white blood cells and bacteria). In the absence of a similar relationship we assume that the source of RNA-se in polio specimens is the cytoplasm of motor neurons

in chromatolysis, because it follows the time sequence of, and may play a part in the disintegration of, the pentosenucleoproteins of the Nissl body.[1, 12, 13, 14] On the other hand, the absence of DNA-se[15] emphasizes that the poliomyelitis virus attacks first of all, if not exclusively, the tigroid substance in the neurons. Experimental data[16] suggest that the PNA fraction is decreased, while the DNA is unchanged, in the CNS of mice infected with the MM strain. Hydén[17] stated that the PNA content of nucleoli increases and clumps of PNA develop in the nuclei of polio-infected nerve cells. These findings supplemented by our observations underline again that only the PNA metabolism is altered during poliomyelitis and therefore the simultaneous increase of RNA-se and absence of DNA-se in the CSF is of special significance. Another possibility would be a toxic factor, affecting one nuclease, leaving intact the other. Bodian and others[16, 18] found some biochemical similarity between experimental chromatolysis and cyanide poisoning. It is interesting to note that the DNA-se is very sensitive to CN compounds and the RNA-se is not.[11]

The relatively higher phosphorus values observed may depend on the simultaneous activity of nucleases, nucleotidases, and phosphatases.[19] We cannot explain the results at pH 5 in some nonparalytic polio specimens. One could surmise that the lack of autolysis of phosphate derivatives in CSF's of polio cases[2, 3] and the different pH optimum in nonparalytic samples may be connected with the resynthesis of the chromatin which tends to utilize all the available material for regeneration. The absence of paralysis indicates that the functional balance is in equilibrium in the motor nerve cells. These assays suggest the existence of two different kinds of RNA-se[20] in some CSF, or at least various pH optima, for the same enzyme.

The patterns observed in the virus group may be found occasionally in other CSF's also, but there is generally a quantitative or a qualitative difference in enzyme activity, as, for instance, in the neurologically normal group; the highest RNA-se value here (a gastroenteritis case) was due to dehydration. Similarly, the frequency and intensity of nuclease activity is different in meningitis specimens, which, in contrast to polio, do contain DNA-se in the majority of cases. Otherwise the presence of RNA-se at a high level and the absence of DNA-se at the same time is rare in diseases other than polio, and is accompanied by signs that make the differential diagnosis clear. Out of 200 fresh or freshly frozen CSF's of polio patients, only one in which growth of *Salmonella typhimurium* was ascertained displayed DNA-se activity.

SUMMARY

Characteristic RNA-se and DNA-se patterns were observed in the CSF of poliomyelitis and other patients with virus disease. These patterns differ qualitatively and quantitatively from the majority of other specimens. The results are statistically significant. With quick methods the two nucleases can be demonstrated within forty minutes. The pathological importance of these findings is discussed.

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New York—On Wednesday the 4th of this Instant, a Boy between the Age of 14 and 15, named John Harvey, who had been afflicted with Stone in the Bladder for upwards of eight years, was cut in the presence of several eminent Gentlemen of the Faculty, by Mr. Richard Bayley, Surgeon of this City. The Stone extracted was of an irregular Oval Figure, two Inches in Length, and three in Circumference.

The Operation (tho' laborious and difficult) was performed with great Judgment and Dexterity, in so much, that in the Space of 36 Hours after the Operation, the Urine begun to pass partly through its natural Passage; and on the 11th Day, the Urine passed off so complete, that not one Drop has escaped through the Wound, so that he is now perfectly recovered.

New York Gazette, January 25, 1773.

PEDIATRIC EVALUATION OF BRAIN-DAMAGED CHILDREN

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IN August, 1952, the prenursery school program for children with cerebral palsy was started at Children's Hospital in San Francisco, sponsored by the local United Cerebral Palsy Association.[1]

For several years members of the hospital staff, medical and ancillary, have participated in a weekly seminar on childhood ecology.[2, 3] This experience furnished the staff with excellent background and preparation for work with the handicapped child. A grant from the national U.C.P.A. in 1954 has made it possible to expand the educational program with special emphasis on the growth and development of normal children. The present report was further stimulated by a concomitant study of nutrition being conducted by Dr. Elizabeth Macaulay under a grant from General Foods.

MATERIAL

Since the opening of the cerebral palsy program August 1, 1952, 131 children have registered. Table I lists the main diagnosis for each child and Table II the secondary diagnoses. It will be noted that the average per patient is 2.3 major handicaps. The greatest number of diagnoses on a single patient was six, consisting of spastic quadriplegia, congenital hip, seizures, strabismus, mental retardation, and microcephaly. In Table III are listed the etiological factors as determined by history and physical findings on one hundred of the cases in which the study was complete enough to make an evaluation.

Several children were seen merely once in consultation and since detailed studies and follow-ups were not always available on these, analysis of various aspects may be based on different figures. This will be indicated in each instance. Of the total number of children registered, seventy-four were males and fifty-seven females. The ages varied from birth to 10 years, with 77 per cent having birth dates in 1950 or later.

Under "delayed motor development" in Table I are included infants and young children who still show only weakness of muscles, such as failure to support head or to sit up. Also included in this group are brain-injured children with mental retardation and delayed muscle development who came to the cerebral palsy clinic primarily because of failure to walk at the usual age. Apart from the above group were a few children of normal intelligence who were merely late walkers, accomplishing it at nearer 2 years than 1. Cases of central nervous system damage without motor involvement included children with speech difficulties, nystagmoid eye movements, and some with autistic or psychopathic symptoms.

The number with seizures, namely, seventeen, appears low. A partial explanation for this figure is that the children are young. Convulsions, recognizable as such, may not have appeared at this early date. In retrospect it was also apparent that the history of an occasional febrile con-

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vulsion may have been missed. The seventeen cases therefore represent children requiring treatment for seizures.

cases where the bone age was taken, ten showed rather marked retardation. This was unrelated to the severity of the physical handicap and, therefore,

TABLE I. PRIMARY DIAGNOSIS

Cerebral palsy—spastic		52
Quadriplegia	24	
Paraplegia	11	
Hemiplegia L	8	
R	7	
Triplegia	2	
Athetoid		11
Ataxic		12
Atonic (flaccid)		11
Tremor		2
Incoordination, motor		5
Delayed or retarded motor development of neurogenic origin		24
Central nervous system damage without motor involvement		8
Normal children—slow development		3
Miscellaneous		3
Total		131

TABLE II. SECONDARY DIAGNOSIS IN SAME 131 CASES

Mental retardation		39
Strabismus		30
Serious eye or visual disturbance		16
Blind	3	
Enucleated eye	1	
Hemanopsia, cortical visual disturbance, etc.	12	
Seizures		17
Speech disturbances, including so-called aphasia		16
Deafness—total		1
Multiple congenital anomalies		13
Congenital heart		5
Congenital or aplastic hips		5
Psychological problems, including autistic and so-called schizophrenia		17
Microcephaly		7
Arrested hydrocephalus		4
Total		131

TABLE III. ETIOLOGICAL FACTORS

Congenital anomalies	30
Complications of pregnancy and delivery at term	25
Prematurity per se and/or with complications	23
Postencephalitis	6
Viral infections, first trimester	5
Kernicterus	4
Twin births with complications	4
Injury—postnatal skull fracture	3
Total	100

PHYSICAL STATUS

The Iowa growth curves were used for evaluation of nutrition. Of ninety-nine children studied by this method 73+ per cent fell on or below the 3 percentile line in height, and 70+ per cent below it in weight. In twenty-two

cannot readily be explained on the basis of nonuse or the failure to exercise normally. Many children with a normal bone age were much more severely handicapped so far as motor activity was concerned.

The teeth, in general, erupted in a fairly normal time and manner, even in the children having a retarded bone age. In the severely spastic, and frequently in the athetoid, the teeth were found to be ground down almost to the gums, and the oral cavity was deformed, usually with a high, narrow palate and a marked overbite. This presumably occurs from the involuntary muscle pulls.

In a sampling of blood counts, the hemoglobin was well maintained. Undoubtedly this is due to the slow weight gain which does not put particular stress on the iron storage.

The picture presented by these children, therefore, is that of small and fragile individuals. They have a poor appetite, vomit frequently; they have many respiratory infections; they perspire a great deal, making the care of the skin particularly difficult. To date, four deaths have occurred in the group. The children recall the experiments of Duraiswami[4] on chick embryos treated with insulin. He describes the chicks as delicate, fragile, and hard to rear. Studies in schizophrenia like those carried out in the Tulane department of psychiatry and neurology[5] would indicate that the major reason for the fragile constitution of these children, as well as the specific pathology such as retarded bone age, are neurogenic in origin. It is tempting to explain the physical retardation on poor appetite, difficulty in eating and swallowing, and various other handicaps, but it is in all probability central in origin and not due to these secondary factors per se. When biochemical and electrophoretic studies are carried out, probably additional constitutional variations on the same basis will be discovered.

INTELLECTUAL STATUS

That brain damage should frequently interfere with normal mental and intellectual development is axiomatic. The severity may depend on the type, extent, and localization of the lesion or lesions. Intelligence tests, modified for the handicapped, have been the subject of much research. The younger the child, the more difficult is the evaluation for the obvious reason that development early is based almost entirely on physical skills of which the brain-damaged child may be totally or partially deprived. Often the educator can better estimate the educability of the child than can the psychologist and the two disciplines should work closely together, as well as with the physician and members of other disciplines such as physiotherapy, occupational therapy, and speech therapy, in order to obtain as accurate an estimate as possible of the child's intellectual capacities. The necessity for early labeling of brain-damaged children with an I.Q. is being abandoned in favor of other methods of interpreting development and capacity. The snap diagnoses of mental deficiency by physicians are still too frequently practiced and often justifies the rage of parents of handicapped children toward professional personnel. Random examples suffice to illustrate the errors made. We have found that a child can vary in I.Q. from 70 to 105 in one year. An athetoid child rated as mentally retarded by competent psychiatric methods at the age of 18 months, at 4 years shows superior ability. In one instance, a child seen by the same group on two successive years was thought to be "obviously retarded" in the first instance and "obviously superior" in the second.

Brain-damaged children follow the same general developmental pattern that normal children do, although they may do it at a slower pace. The 2-year-old is negativistic, dependent on his mother, and individualistic in his play. The same child at 3 is more eager to enter into play with the group; he has graduated somewhat from his dependence and is far more cooperative. He may suddenly show

skills that it had been assumed were beyond his capacity. There is no justification for condemning these children during the first five years of life to the classification of mental defectives. It is better to expose them to every opportunity and stimulus possible and judge their capacities later. If this is not done, the unfavorable environment created by pessimistic attitudes may be responsible for failure to attain inherent potentialities. It should also be noted that in our experience children with pneumoencephalograms showing cortical atrophy early in life may later have normal studies and show surprisingly good development.

EMOTIONAL STATUS

It is generally accepted that human beings have a threshold to stress and strain that varies greatly, but no one is invulnerable to a breaking point. Our military personnel probably furnished the best recent examples. It is assumed, though undoubtedly erroneously so, that infants are immune to similar breaks under stress. The emotional environment that these children are subjected to are exceedingly pathological. Frequently the most tender parents forget to play with the child and talk to him, simply because the child is incapable of responding in the customary manner. In addition, the child is often hospitalized and painful procedures are carried out. Children have pneumoencephalograms, craniotomies, eye operations, feet or hip casts, and numerous other, often multiple, such experiences. It is reasonable to wonder how much this may contribute to mutism, to autism, or to the schizoid type of child one sees in the group. Less attention to I.Q.'s and more to healthy environment should be the concern of the personnel in contact with these children, especially so during the first five years of life. Their emotions certainly call for as careful nursing as their physiques.

SOCIOECONOMIC PROBLEMS

The handicapped child usually presents the parents with a financial burden which cannot be met except possibly by the very wealthy. The multiple handicap nature of the disease requires not only one consultation, but repeated visits to many doctors and other professional personnel. In addition to the pediatrician, he will usually be seen by an orthopedist, a neurologist, a neurosurgeon, or both, an ophthalmologist, frequently by an otologist and speech therapist. More than average hospitalization is required, and expensive procedures such as electroencephalograms, x-rays, and braces are usually necessary. Even care in the home is greater and often more expensive than for the normal child. Special baby sitters at a higher rate are often indicated. The mother is never as free to take the child out in the car or in the stroller as she is with the normal. Everything about him adds to the expense of care. Frequently, insurance is not available to these children. The families are in need of a realistic approach toward help in meeting the real and justifiable demands of the handicapped child. The numerous organizations, parent sponsored, that are appearing is evidence of the reality of this need.

THERAPY APPROACH

The first essential for therapy is an accurate medical diagnosis. Cerebral palsy almost more than any other

disease requires teamwork. In this team the pediatrician should be in charge of coordinating the diagnostic and therapeutic programs and interpreting the results to the parents. It is important to establish as nearly as possible the site and type of the main lesion as well as the functional by-products of this. The motor handicap is usually the most readily determined, the others are more difficult. If vision is not normal, how is it affected? Is it ocular or central or both in origin? Hearing may be present, but is the range normal? Is there present any type of aphasia? What is the child's real intellectual capacity? Is it obscured by either his physical or his emotional handicap? After the diagnostic study, it is necessary to have a realistic working estimate of the child's ability. Roughly, this can be classified as (1) possible restoration to normal, (2) limited, but self-sufficient eventually, (3) partly dependent always, (4) probably totally dependent always. This should be a working classification, and reevaluated at intervals of three, six, or twelve months, depending on the age of the patient and the progress made. After the child has been thus classified, all rehabilitation efforts should be directed toward the goal. In the young child, primarily under 3 or 3½, much of therapy must be done in a play situation. This is the great advantage of the nursery school, for in addition to being able to accomplish exercises and corrective measures, the child benefits tremendously socially from this group work. Probably, finally, socialization will be the greatest therapy the child has experienced of practical value for his future development. This is particularly true if parent education is accomplished at the same time and family adjustment can be established.

SUMMARY

From a pediatric point of view, brain-damaged children are small, fragile, and subject to frequent infections.

The intellectual capacity of these children is difficult to determine because of physical handicaps. Final judgment should be postponed and the parents encouraged and instructed in techniques to stimulate the interest of the child.

The children should be guarded against unnecessary traumatic procedures and separation from mother even more than normal children, lest such exposure superimpose on the child psychopathological damage.

The cost of adequate care of the brain-damaged child is great and frequently cannot be adequately met by private means. Diagnostic studies and therapy should be done by qualified teams and every effort should be made to rehabilitate the child and to support the parents in their attempt to do this.

The nursery school situation is a valuable therapeutic adjunct to the life of the young handicapped child.

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GRASS INFLORESCENCES AS FOREIGN BODIES IN THE RESPIRATORY TRACT

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THREE children have been observed here in recent years following the aspiration of inflorescences or spikes from the common timothy or foxtail grasses. They have exhibited many features in common while deviating, as a group, from the course usually expected of children with the more ordinary respiratory tract foreign bodies.

CASE REPORTS

CASE 1.—The first child was 4 years old when seen in October, 1946, because of hemoptysis which had been present for nine days. During the preceding July he had been hospitalized in another city, with cough, chest pain, and fever which had persisted in variable severity despite the usual gamut of treatment. Dullness and diminished intensity of breath sounds were noted over the region of the right lower lobe at the time of his first contact here and, roentgenologically, "infiltration in the lower third of the right lung . . . having the appearance of a low-grade bronchopneumonia" was reported (Fig. 1). Continued cough, accompanied by sufficient blood loss to require replacement, led to bronchoscopy soon after admission. No foreign substance was visualized, but bronchiectatic changes confined to the lower, medial, posterior segment were demonstrated. No more hemoptysis occurred during the following year, but only rarely was he free from cough and minor elevations in temperature. Films were obtained periodically and no essential change from the original pattern was discerned, so bronchoscopy was repeated twelve months after the initial symptoms and a well-preserved spike of foxtail, measuring over 2 inches in length, was removed. Since then, he has developed in an orderly manner and has had no symptoms referable to the chest, although films as late as six years following the incident continue to show a segmental area of infiltration which undoubtedly represents permanent bronchiectasis and fibrosis.

CASE 2.—The second case, a girl, 18 months of age, was seen in July, 1947, with paroxysmal cough and fever for three weeks. She had received the inevitable penicillin injections without benefit, and examination revealed dullness and decreased intensity of breath sounds over the right base. Films demonstrated "a dense infiltration in the base of the right lung having the appearance of an inflammatory consolidation" (Fig. 2). Initial bronchoscopic examination was not fruitful but, when her symptoms persisted, another effort was made after two months and a spike of timothy grass was removed piecemeal. For a period following this, she responded rather strenuously to minor respiratory infections and, on several such occasions, developed increased infiltration in the area of original damage. Over the past four years this tendency has gradually diminished while she has fulfilled in growth the pattern suggested by her record prior to the accident. A small segment of bronchiectasis and fibrosis is visible in recent films so that her situation is reviewed periodically, but little basis for intervention has been defined.

CASE 3.—In July, 1949, the third child was seen at 22 months of age because of fever for twenty-four hours.

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He alone of the three had been noticed to place something in his mouth and, while the parents were most concerned with the possibility of food poisoning, the admitting physician raised the question of a pulmonary foreign body. This was entertained more seriously when a paroxysmal cough developed

Fig. 1.

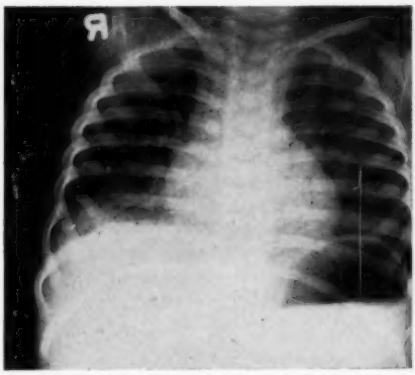

Fig. 2.

Fig. 3.

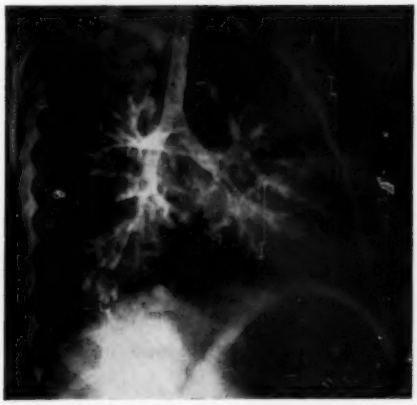

Fig. 4.

on the following day and the roentgenologist reported "a dense infiltration at the base of the right lung having the appearance of an acute inflammatory consolidation associated with a tendency to atelectasis" (Fig. 3). Treatment with sulfadiazine and penicillin during the ensuing week altered neither the clinical nor the roentgenologic findings, and bronchoscopy was performed. Purulent secretions were removed from the right bronchus but no foreign body was encountered. Cough and x-ray evidence of pneumonitis persisted during the remainder of the summer and early fall, so a second bronchoscopy was performed, still without visualization of an offender. There was little change in either clinical or roentgenologic findings through the following year, so a third bronchoscopic examination was done in

October, 1950, and, again, nothing of note was accomplished except the demonstration of bronchiectatic changes in the posteromedial segment (Fig. 4). He developed, following this last instrumentation, a purulent meningitis from which immediate recovery ensued with adequate chemotherapy. Another period, this time of eighteen months, elapsed with no essential change in status; he continued to cough, experienced periodic elevations in temperature, failed to grow at the expected rate, and demonstrated persistent radiologic changes. Finally, late in 1952, lobectomy was performed by Dr. Jack Hertzler, and buried deeply within the removed tissue was a fully preserved spike of timothy grass, surrounded by an inflammatory and fibrous reaction. This boy has been free from symptoms since surgery and has recovered the ground in growth and development lost during his prolonged illness.

DISCUSSION

It is noteworthy that each child became ill in July when these native grasses reach fruition and when the small child is outside unattended for his play. Each case was assumed to represent an inflammatory pneumonitis of the right lower lobe and was so treated for variable periods. The physical nature of the inflorescences explains many features of the subsequent course. Lack of denseness precludes either complete or ball-valve bronchial obstruction and the development of massive atelectasis or emphysema. The arrangement of spikelets in both species imparts an element of self-propulsion, and, thus, limited migration toward the lung periphery and beyond the range of easy bronchoscopic visualization. However, lack of extreme rigidity in both species during the July phase of maturity prevents continued penetration into the chest wall as has been observed by Sehring and Shaw[1] with a sturdier western species. In none of our patients was there evidence of marked chemical irritation or hypersensitivity to the foreign protein so that continuing violent response was absent, in contrast to what we so often see after aspiration of nuts. Finally, it is interesting to note the persistence of segmental bronchiectasis and fibrosis without detriment to continued good health and normal development in two of these children, a point made earlier by Jones and associates[2] for the permanent bronchial changes following occlusion due to tuberculosis.

SUMMARY

The course of three children who aspirated into their lungs inflorescences of common grasses is described. Each became ill in July, with inflammatory changes in the right lower lobe; initial bronchoscopy in each was unproductive, but in two the offending objects were removed at subsequent instrumentation. With the third child, lobectomy was performed and the spike was recovered from the surgical specimen. The two children in which bronchoscopic removal was successful have continued to reveal x-ray evidence of segmental bronchiectasis and fibrosis, but each has remained well and the need for surgery has not become apparent.

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COARCTATION OF THE AORTA IN SIBLINGS

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IN RECENT years, the familial occurrence of congenital heart defects has been recognized with increasing frequency. On rare occasions, identical malformations have been detected in two or more members of the same family. The relative infrequency of such reports, however, has served as a stimulus for this publication.

CASE REPORTS

CASE 1.—R. M., an 8-year-old male, was first seen in July, 1947, with complaints of frequent severe muscular cramps in both legs for the preceding four years. There was a history of a heart murmur having been present since early infancy, but no restrictions had ever been placed upon his physical activities. No episodes of cyanosis or exercise intolerance had been noted.

The family history revealed that a 1-year-old brother was under treatment for bilateral congenital cataracts. There were no other siblings. The mother and father were living and in good health.

A thorough search was made into the early gestational history of both pregnancies. There was no history of acute viral or bacterial disease, of chronic constitutional disease, or of exposure to toxic agents or roentgen rays. The maternal diet was adequate and supplemented with vitamins. Conception was achieved with ease and there were no abortions or threatened abortions. Both pregnancies terminated spontaneously at term.

Physical examination of the patient revealed classical signs of coaretation of the aorta. A grade III systolic murmur was audible over the base of the heart and in the suprasternal notch, and there was a pronounced systolic thrill in the latter area. Visible pulsations were noted suprasternally and in the posterior intercostal spaces. The radial pulse was strong, but femoral pulsations could not be palpated. The blood pressure reading was 156/100 mm. Hg in the right upper extremity and zero in the lower extremities. On the basis of these findings, a diagnosis of coarctation of the aorta was made and surgical repair was performed on Sept. 24, 1947.

*Surgical Findings**.—"The coaretation was seen in the usual position just below the ligament of the ductus arteriosus. The second and third intercostal arteries were greatly dilated. The subclavian was about normal size but the common carotid came off just proximal to the subclavian and it was greatly dilated and bifurcated about 1 cm. from the arch of the aorta into its two large branches, the internal and external carotids. The common carotid was so dilated as to partially overlap the subclavian."

Postoperative Course.—The course following surgery was uneventful. Leg pains disappeared promptly and femoral pulsations and blood pressures could easily be obtained. The child was discharged after a short hospital stay and was soon returned

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*Courtesy Dr. John Jones, Los Angeles.

to full activity. He has had no symptoms referable to the cardiovascular system. He was last examined on Sept. 23, 1954, and the cardiac auscultatory findings were unchanged. However, femoral pulsations were normal and the blood pressures of the right upper and lower extremities were 140/72 and 180/140 mm. Hg, respectively.

CASE 2.—C. M., female, born Aug. 4, 1948, was the third child of this family. Again, the gestational history was noncontributory. During the first year, the patient was examined at monthly intervals. No abnormalities were noted. In September, 1950, a tonsillectomy was performed because of frequent tonsillar infections. In July, 1951, she was examined because of severe muscular cramps in both lower extremities for two months. It was at this time that a grade I systolic murmur was audible for the first time over the base of the heart. The murmur was transmitted to the neck vessels on both sides. The radial pulse was strong but the femoral pulsations could not be palpated. The blood pressure reading was 180/110 mm. Hg in the right upper extremity and zero in the lower extremities. The findings indicated that this child also had coaretation of the aorta, and surgical repair was performed on July 8, 1953.

*Surgical Findings**.—"The usual findings of a coaretation at the ligamentum arteriosus 2 cm. below the left subelavian artery were noted. The opening in the coaretation was 1 mm. in diameter on the inside. The intercostal arteries were dilated below the coaretation."

Postoperative Course.—The immediate postsurgical course was uneventful. Leg pains disappeared promptly. Early in the postoperative period, femoral pulsations and blood pressures could be obtained. The child was discharged and soon returned to full physical activity. Five months following surgery, she contracted paralytic poliomyelitis with extensive involvement of the muscles of all four extremities, of the back, and of the chest. She was placed in a respirator for five months. Her cardiac status was last evaluated on Aug. 3, 1954. At that time, she was then fully ambulatory but was under orthopedic observation for residual paresis of the intercostal and spinal musculature. The cardiac auscultatory findings were the same as those noted preoperatively. However, femoral pulsations could be palpated and blood pressures in the right upper and lower extremities were 104/42 and 110/50 mm. Hg, respectively.

*Courtesy Dr. John Jones, Los Angeles.

COMMENT

Congenital cardiac malformations occurring in more than one member of a family are not unusual. Eastman[1] states that if a malformation is present in one offspring there is a 20 per cent chance that there will be some abnormality in subsequent offspring of the same mating. He further states that there is a 2 per cent chance that the abnormalities will be identical. Results of clinical investigations by Campbell[2] suggest a significant increase in the incidence of congenital cardiac defects in relatives of children so affected. Recent clinical studies by McKeown and associates[3] indicate that there is a sixfold increase in cardiac abnormalities among the siblings of children with congenital heart disease.

Little is known of the familial incidence of specific types of cardiovascular abnormalities. From the studies of Anderson,[4] it can be stated that there is a definite tendency for patent ductus arteriosus to recur among siblings. To what extent this is true of other cardiac malformations has not yet been established.

Only three reports of familial coarctation of the aorta have appeared in the literature. Walker[5] described the occurrence in a father and son. Klemola[6] reported two families, each of which had two brothers with coarctation of the aorta. More recently, Taylor and Pollock[7] described the condition in a mother, son, and daughter. McKeown and associates[3] found no recurrence of coarctation of the aorta in twenty sibs of twenty-two patients.

With the continued progress in the diagnosis, prophylaxis, and therapy of acute rheumatic fever, one may anticipate a steady decline in the incidence of rheumatic heart disease. On the other hand, our present management of infants and children with congenital heart defects has made it possible for many of them to reach the childbearing age. A relative increase of congenital heart disease may then be expected if heredity is an etiological factor. The causative factors of congenital cardiovascular defects, however, continue to remain primarily a matter of speculation. The recognition of such defects as complications of maternal rubella suggests the possibility that other viruses may exert a similar influence. The increasing recognition of the familial occurrence of congenital heart disease suggests that genetic factors may also be involved. If so, they are probably of a recessive type since secondary cases in sibs are more frequent than in parents and children.

Information concerning the fundamental causes and pathogenesis of congenital heart defects is trailing far behind the rapidly accruing knowledge of diagnosis and treatment. Complete solution of the problem of congenital cardiac abnormalities awaits a more thorough understanding of the etiological factors involved.

SUMMARY

Coarctation of the aorta in two siblings is described. A third sib has bilateral congenital cataracts.

The etiology of congenital heart disease with emphasis on its familial occurrence is briefly discussed.

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LIBERAL VISITING POLICIES FOR CHILDREN IN HOSPITALS

A Report by
The Citizens' Committee on Children of
New York City, Inc.

EARLY in 1954 the Citizens' Committee on Children of New York City, an organization of lay and professional persons interested in the quality and quantity of the City's services for children, undertook to explore the frequency and length of visiting hours on children's wards of New York City's hospitals, both public and voluntary. The CCC has from time to time looked into many aspects of health, education, and welfare facilities in order to inform itself and the community about needs and resources. A hospital's relationship to its young patients and their parents was considered of sufficient importance to warrant attention.

It seems reasonable that services for children should be designed with due consideration for the family unit. When a child is sick, his need for the comfort and support of his parents is even greater than usual, and when he enters a hospital the need can be almost overwhelming. It therefore seems wise that parents should be with their child, to comfort him by their presence and be reassured about his progress.

Obviously a hospital's basic philosophy toward its patients and their families is reflected in its routine procedures. For example, in procedures governing admission, one hospital may require parents to leave as soon as the necessary papers are signed and the child is removed from the admitting office. Another may help the parents prepare their child for hospitalization and then permit them to remain with him until he and they have become acquainted with the ward, with some of the other children, and, most important, with the new people who will be taking care of him. The hospital philosophy is also reflected in the policies governing surgical procedures; one hospital may deny parents access to the child immediately before or after operation; another may encourage parents to remain with their child until he is taken to the operating room and to be waiting for him when he returns. There are many critical points in a child's hospital experience in which the parents can be encouraged to participate or from which they may be excluded.

A hospital's visiting hours are still another manifestation of its general attitude. It was assumed that New York City would reflect the nationwide trend toward liberalization of visiting privileges for parents, but the nature or extent of the local trend was unknown.

The Health Section of the CCC undertook this exploration not as a comprehensive study but as a survey from which to gain an impression of the current situation and evidence

of change, actual or potential. It was hoped, too, that through visits to hospitals and discussion of what had been learned in other hospitals, any local trend toward more flexible schedules might be stimulated and hastened.

Specifically the study was designed to determine whether more detailed information about visiting schedules is needed as a prelude to an intensified educational effort to improve the policies which visiting schedules reflect.

The approach to this project had four parts which, except for the first, were carried on concurrently:

A telephone survey of the seventy-five member hospitals of the Greater New York Hospital Association which have pediatric beds was conducted. The hospitals were asked for information as to visiting hours on children's wards and the number of pediatric beds in each.

Visits were made to selected hospitals to learn how their liberalized schedules had come about and how they were working out.

Letters were sent to hospitals in other cities and countries to learn of and benefit from their experience.

Discussions were held with pediatric and nursing leaders in New York City and elsewhere offering support in their efforts to improve hospital care of children.

The results of the telephone survey on visiting hours are shown in Table I. In February, 1954, 60 per cent of the hospitals, with 72 per cent of all pediatric beds, reported that parents were allowed to visit no more than three times per week. In contrast to the twenty-five hospitals with 825 beds which permitted daily visiting, twenty-six hospitals with 1,694 beds allowed parents to visit no more than twice a week. Moreover, hospitals which allowed more frequent visiting had, for the most part, more flexible hours, whereas those with limited schedules tended to retain the time-honored 2:00 to 3:00 P.M. or 3:00 to 4:00 P.M. hours which are seldom convenient for fathers who work or mothers who have other children coming home from school at 3:00 P.M.

TABLE I.—VISITING HOURS

NUMBER OF VISITING DAYS PER WEEK	NUMBER OF HOSPITALS	NUMBER OF BEDS
7	24 (32%)	825 (23%)
5	2 (3%)	47 (1%)
4	4 (5%)	129 (4%)
3	19 (25%)	971 (26%)
2	23 (31%)	1,314 (36%)
1	3 (4%)	380 (10%)
Total	75 (100%)	3,666 (100%)

The telephone survey was substantially correct in February, but the situation is changing rapidly. Many of the hospitals with the most liberal programs had established them within a matter of weeks, while others were planning new schedules to go into effect within the month.

There is much talk about more flexible visiting hours and much experimentation. Many pediatric and nursing leaders are eager to see a more rapid and widespread acceptance of the role of the parent in helping a child get well.

To date about a third of the City's hospitals having the most liberal

schedules have been visited. Communications have been received from hospital personnel in a dozen cities as well as from the Ministry of Health in Britain. Key members of leading pediatric and nursing organizations and educators in both fields have been interviewed.

Fairly general agreement was found on certain obstacles which had to be overcome before more flexible visiting schedules could be established. Some had anticipated more obstacles than were actually encountered, while others had failed to take into account all the difficulties which lay ahead. But, with few exceptions, hospital personnel were pleased with the more liberal visiting privileges for parents and ready to deal with problems as they might arise.

The following are some of the more common questions and problems reported by the persons interviewed:

1. *Physical Plant.*

Many hospital buildings are old; fire hazards must be guarded against; elevators are of ancient design; beds are crowded together with little room for visitors. It was found, however, that there is less strain on the facilities of an old building if visitors can come and go over a period of several hours and are not herded together during one peak period—less strain on the elevators, less danger of panic, and less crowding of the wards because not all parents are on the ward at the same time.

2. *Fear of Cross-Infection.*

The availability of antibiotics has greatly lessened the hazard of cross-infection and has given hospital personnel the courage to experiment. Their experience has demonstrated that the increased hazard is minimal.

3. *Parents Might Abuse the Privilege.*

Most hospital personnel have encountered the demanding parent who feels that his child is not receiving all the attention he should have, or the anxious parent who buttonholes staff in corridors to plead for reassurance and answers to questions that have already been answered many times. The parent who brings forbidden food to a child on a special diet or the mother who upsets a hard-won nurse-patient relationship by her critical remarks to the child about the nurse—these are only some of the more annoying categories of parents who disturb the orderly process of hospital routine. When hospital staff contemplates more frequent and longer visiting hours, it is inevitable that these more troublesome parents should come to mind with the result that more liberal visiting schedules tend to be equated with increased exposure to problem parents.

The general experience has been that parents have not abused the privilege of more frequent and longer visiting hours. A small number of parents will cause trouble regardless of hospital rules and regulations. Some of our informants indicated that more flexible schedules and a firm, consistent approach to the disturbed parent's demands have actually resulted in less rather than more trouble on the ward. It was generally agreed that parents have proved more adaptable and cooperative than had been anticipated. Parents appreciate the opportunity to spend more time with their children, and good will toward the hospital has increased proportionately. One hospital reported that payment of bills had been stepped up and autopsy consents increased.

In several hospitals Puerto Rican parents were observed sitting quietly by their children's beds, so quietly that at first one tended not to notice that they were there. From staff comments about the gentle, passive,

self-effacing manner of Puerto Rican parents we gained the impression that the high proportion of such patients (as much as 85 per cent in one large hospital) may have helped the hospital personnel to accept more frequent and longer visiting hours.

4. *Shortage of Personnel.*

Since the infrequent one-hour visiting schedule of the past was usually preceded by a flurry of cleaning up the ward, getting the children into clean gowns, and keeping them tidy for the arrival of the parents, it was always a strain on nursing personnel. The arrival of the parents often precipitated crying, hyperactivity, and general confusion, and their departure was usually followed by even more crying, some vomiting, and generalized anxiety that was characterized by restlessness, loss of appetite, and sleep disturbance. In view of the shortage of nurses and auxiliary personnel, it was feared that more frequent visiting would keep the ward in turmoil, place an added burden on staff, and make it more difficult to get or keep nurses.

This has been found to be untrue, however, in hospitals where liberal visiting hours are a reflection of a positive attitude toward patient and parents. Because the parents come and go freely, there is no peak period and no artificially stimulated anxiety; and the children quickly learn that they can believe their parents when they say that they will be back soon. Where parents are expected to help feed the children and get them ready for bed, it has relieved nursing personnel of routine tasks and freed them for other duties. This is especially true on the toddlers' wards in hospitals which subscribe to the theory that patients should be out of bed as much as possible. The average, inquisitive 3-year-old child requires considerably more supervision than a busy floor nurse can give.

The important contribution of volunteers was stressed in many interviews and letters. The chief of one pediatric service commented, "Without our volunteers we could not run our children's service." The regular attendance of carefully selected and trained volunteers is apparently taken for granted in many hospitals.

5. *Responsibilities of a Teaching Hospital.*

A teaching hospital must organize its ward schedules to facilitate the optimal use of teaching personnel and the most efficient allocation of student time. It was feared that if parents were to be allowed to visit freely, their presence on the ward would interfere with teaching rounds, ward rounds, treatment procedures, and other necessary aspects of hospital routine.

While philosophy and practices differ from hospital to hospital, there is considerable agreement that these fears are not justified. Some hospitals begin visiting hours at noon by which time rounds are completed. Some ask parents to leave the ward during rounds or treatment procedures, others do not. In hospitals where parents are permitted to remain, they have been found useful for the additional information they can provide as well as for the beneficial results of their increased understanding of the patient's illness and treatment. Furthermore, there is agreement on the value to residents and nursing students of the increased availability of parents on pediatric wards. As one chief of service put it, "It helps me to teach the residents that a pediatrician's practice is composed of the parents of his patients." A pediatric nursing instructor also pointed out that the new regimen helps to break down the student nurse's isolation from the community. Another pediatric educator commented that "When the parents aren't around every day, the young doctor and nurse get a very false idea of the child. Children live with families, but it's easy to forget that in a hospital which keeps parents at a distance."

6. *The Autonomy of the Services.*

Procedural changes in a large hospital with separately administered pediatric services—medicine, general surgery, eye, ear, nose and throat, orthopedics, etc.—present many practical problems. The harried administrator of the over-all institution has many conflicting demands on his time and energy. If one of the pediatric services wishes to change its procedures and all or most of the others are lukewarm or even cold to the idea, the administrator is unlikely to look favorably on the suggestion. If the individual specialty can make the change without disturbing the traditional over-all pattern, it may be permitted. Or if the individual specialty can persuade the others, there is more likelihood of its acceptance.

It was agreed that a policy of change of this kind cannot and should not be foisted on other groups who do not accept the validity of the idea.

This problem has been handled in various ways and at least partially solved by many of the hospitals. It would appear that the more common practice is to change the policies within the individual service, usually the medical service, and attempt by example and discussion to persuade the others. Surgical services seem to be somewhat less inclined to liberalize visiting, and on the basis of our limited information it would appear that orthopedic services have even stronger reservations. On the other hand, in one hospital with unlimited daily visiting, parents of polio patients are taught physiotherapy procedures so that they may carry on their child's treatment on the physiotherapist's day off.

7. *Inertia.*

Last but not least is the inertia which pervades all institutions. The tendency to perpetuate old practices, merely because things have always been done that way, is not unique to hospitals. But in the hospitals as elsewhere it is a passive influence against change that is often more difficult to fight than active resistance. It is more insidious and less tangible than the skeptical "show me" attitude of the interested but unconvinced.

Professional organizations have made some headway toward encouraging more widespread review of current practices through journals, meetings, workshops, and other devices designed to stimulate interest and spark the potential leadership in individual hospitals. Young residents with sufficient enthusiasm have aroused their tradition-bound colleagues. Nurses with experience in more liberal settings have broken through the wall of inertia in some hospitals. Social workers and volunteers have helped stimulate interest and action. There is no precise formula but much evidence that it can be done. And there is also the factor of rivalry—publicity for one hospital's achievements may arouse others to do likewise. Furthermore, experience has shown that changes need not be drastic. Revision of visiting policies can be undertaken and tested a step at a time—ward by ward or floor by floor. And within a hospital itself, the publicity factor has been seen to operate in stimulating wider application of a new approach.

So far we have noted some of the more positive aspects of liberalized visiting—answers to questions about the feasibility of permitting parents to visit more frequently. By and large these are the beliefs held in hospitals which are convinced of the necessity for protecting and strengthening parent-child relationships during hospitalization. Where the staff believes in that concept, visiting hours reflect the basic attitude which is generally warm, accepting, and supportive.

Some difference of opinion exists among those who are experimenting with daily visiting. These differences were largely concerned with:

A. *Length of Visiting Hours and Their Distribution.*—Hospitals in New York City which have recently liberalized their schedules tend to allow parents to remain for several hours each day—as long as eight hours in some cases. On the other hand, a number of hospitals in other cities with longer experience now average one to two hours daily, some of them reporting that they tried longer periods at first but found that shorter though still frequent visits are more satisfactory. Problems about the effect of long or unlimited visiting hours are numerous, among them:

1. The mother's neglect of household responsibilities and her other children;
2. Strained relationships growing out of the mother's feeling that she must stay with her child throughout the visiting period even though she might wish to be elsewhere. As noted earlier, this has led some hospitals to cut down the stipulated hours as a protection for parents. On the other hand, if the nurses are alert to the potential danger, it can be handled easily. Each mother is asked about her other responsibilities and encouraged to adjust her visiting to dovetail and not overlap. If her own needs drive her to come more often and stay longer than is reasonable, the social worker tries to help her deal with the problem.

The scheduled time of daily visiting varies from one hour at noon, to all afternoon, to two hours at lunchtime and again at suppertime. It depends on the philosophy of the hospital about permitting or expecting mothers to feed their children, and on this opinion differs. It also depends on the hospital's thinking about the percentage of staff that should be available during visiting hours. Some believe that the full nursing staff should be on hand and that a resident should be in attendance to answer questions. Others hold that if the parents feel they are accepted as part of the treatment team, they can be used freely and will quickly become acclimated to the ward and its personnel with no need for special arrangements.

B. *Children's Reactions.*—There was general agreement that more liberal visiting schedules are better for children. At any rate the stormy sessions that used to follow the old visiting hours are absent, although some nurses believe that children become overtired if parents remain for several hours at a time. Opinion differs on what happens to unvisited children when other children's parents are on the ward every day. Some feel that it is an added burden for the unvisited; others hold that in the absence of the frenzied peak periods of former years—something like mail call for overseas troops—the burden on the unvisited is lessensed rather than increased. It is also reported that by the judicious use of a volunteer, the lonely, unvisited child can often be comforted. Furthermore, some hospitals have found that parents tend to visit other children whom they get to know and that one mother often substitutes for another who cannot visit for a time. As one pediatrician remarked, "Parents become neighbors for the duration."

A fairly universal problem is the lack of frequent communication among hospitals in the same city. Hospital people know much more about what is going on in Cincinnati or Boston than about what the hos-

pital in the next block is doing. Some way should be devised to maintain a steady flow of information to all the hospitals in the City. This does not relate to professional data which are available in professional journals but to the practical day-to-day developments and experiences that each hospital must now work out for itself with great duplication of time and effort. It was agreed that experimentation is good and uniformity is not always desirable but that there should be some device by which the necessity for repeating the other fellow's mistakes might be reduced to a minimum.

There is great interest and readiness to experiment with liberal and flexible visiting hours. A number of attending pediatricians who said of themselves, "If you'd asked me about this a couple of years ago, I'd have opposed it," reported a growing enthusiasm for the easier atmosphere of the wards, happier children, and more cooperative parents. While some nurses still had questions, the majority were strongly in favor of the more liberal schedules and talked of new adventures in making the hospital a less awesome place for children and their parents.

The increasing satisfaction which staff members gain from this warmer, more accepting approach to parents seemed a tangible result of these new programs. The old barriers between those who serve children and the parents of the children whom they serve are being whittled down. Nurses, pediatricians, and social workers who recognize this express the hope that as parents are successfully drawn into any one part of hospital routine, other procedures may also be favorably affected.

There is no one pattern in hospitals for the leadership which brings about change. It may be an enthusiastic young resident who breaks through institutional inertia and carries the older members of the staff along. It may be the director of nurses, the pediatric nursing instructor, the chief pediatric nurse of the university nursing school. It may be the chief of pediatrics, or the psychiatric consultant, or the chief of social service, or the director of volunteers, or any combination of these. There are hospitals which illustrate the skillful work of each of these categories of personnel working alone or together with others to bring about a change in official hospital policy.

With enough support inside the hospital and in the community, there is reason to believe that ultimately all hospitals will adopt procedures which reflect their acceptance of the parent as a key figure in his child's physical and emotional well-being, from admission to discharge—or better still—pre-admission to post-discharge.

Medical Progress

VIRAL HEPATITIS AND ITS RISK FROM BLOOD AND PLASMA TRANSFUSIONS

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VIRAL HEPATITIS, with or without jaundice, formerly was comparatively rare, but now is all too frequent. Fifty per cent more cases were reported in 1954 than in 1953.[1] Formerly known as catarrhal jaundice, it must be differentiated from physiologic, obstructive, syphilitic, and hemolytic jaundice, icterus gravis, erythroblastosis fetalis, and toxic hepatitis due to Atabrine, bismuth, INH, Methimazole, TNT, etc.,[2] as well as those related to infections with herpes simplex, herpetic gingivostomatitis,[3] infectious mononucleosis, malaria,[4] and Weil's leptospirosis.

Viral hepatitis occurs in two forms: (a) *epidemic or infectious hepatitis,* which usually is spread by the fecal-finger-mouth route, but occasionally by water,[5] milk,[6] other foods,[7] flies, salivary droplets, and blood. It is more frequent in the winter, and often is preceded by a sore throat for four to five days. (b) *Homologous serum hepatitis* is transmitted by blood or plasma transfusions (especially pooled plasma),[8] human thrombin,[9] unautoclaved syringes,[10] needles, cuts, blood counting lancets and stylets, surgical instruments, etc.; 0.00004 c.c. of infected serum can transmit the disease.[11] It has no seasonal variation. No present method satisfactorily destroys the hepatitis virus in plasma and blood without ruining the usefulness of the blood or plasma. Storing liquid plasma at room temperature for three to six months may reduce the risk but it also may prolong the incubation period so that the serum hepatitis is overlooked.[12] The chance of infection and pyrogens also is increased by the storage of liquid plasma. Viral hepatitis is not transmitted by human serum albumin[13] or gamma globulin. In fact, gamma globulin will prevent epidemic hepatitis,[14] but not the serum variety.[15] Both types of hepatitis are due to viruses (virus A in epidemic and virus B in serum hepatitis). These viruses probably are different as they do not cross-immunize.[16] Virus A, but not virus B, occurs in the stools for five to sixteen months.[17] Virus B, but not virus A, can be found in serum for as long as five years.[17] The incubation period is seven to thirty-eight days in epidemic hepatitis, and two to six months in homologous serum jaundice. Experimental work is handicapped because the disease cannot be produced in laboratory animals except in birds.[18] Because of the possibility of sequelae, human volunteers are scarce.

Incidence.—Eighteen per cent of the 2,322 liver biopsy specimens sent

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to the Armed Forces Institute of Pathology indicated viral hepatitis, the ratio of epidemic to serum hepatitis being 4:1.[19] Epidemic hepatitis may affect 50 per cent of a population.[20] The disease is most frequent between the ages of 4 and 14 years, probably due to contacts in kindergartens and schools.[21] However, an epidemic occurred in an orphanage nursery,[22] and congenital infections may resemble biliary atresia.[23] Second cases in a family are frequent unless 0.1 c.c. gamma globulin per pound is given to the contacts, i.e., the disease developed in 18 of 51 uninoculated exposed children in contrast to 1 in 55 who had had gamma globulin.[24]

Homologous serum hepatitis is an iatrogenic disease, i.e., reports indicate one case of serum hepatitis per 350 single blood transfusions,[25] one to two cases per 100 patients who have received multiple blood transfusions,[26] 1.3 per cent in those who have received small pools of plasma, 11.9 per cent if the pool of plasma had been large,[27] and 20 per cent if the donor had had hepatitis.[28] Serum hepatitis occurred in many of the wounded in Korea who had had blood or plasma transfusions.[29] These percentages would be even higher if they included the mild, nonicteric, and frequently undiagnosed cases (38 per cent of proved hepatitis cases). From the reverse point of view, a significant number of hepatitis patients have had injections of blood or plasma five weeks to five months prior to the onset.[27] Seventy diabetics acquired hepatitis from boiled unautoclaved syringes.[11] These figures indicate a high incidence of carriers of the virus of hepatitis in the population—usually at least 6 per cent,[25] but higher in Italy, Germany, and Korea.

The *onset* in both types may be gradual or sudden, with anorexia, nausea, diarrhea (occasionally), chilliness, fever, malaise, prostration, upper abdominal and generalized pain, gaseous distention, eructation, erythema of the palms and soles, headache, and sometimes eye pain, photophobia, blurred vision, neck and muscular rigidity, numbness, positive Kernig's sign, reduced or increased reflexes, bladder paralysis, peripheral neuritis, vertigo, fatigue, ascites (18 per cent). Neuropsychiatric signs,[30] increased spinal fluid cells (may resemble encephalitis, meningoencephalitis,[31] and poliomyelitis), increased mononuclears (may resemble infectious mononucleosis);[32] distaste for smoking (in adults); followed in five days (12 per cent) to three weeks (50 per cent) by jaundice, enlarged and tender liver. Infectious hepatitis in infants and young children, in contrast to the disease in adults, is not usually characterized by jaundice.[21] As mentioned above, 38 per cent of the hepatitis patients do not have jaundice, and may escape diagnosis. Though it is difficult to distinguish clinically the infectious (A) type from the serum (B) variety, the former usually has a more abrupt onset with nausea and fever, while in the latter, the onset is more insidious and the course is more protracted.[21]

Laboratory.—Bile usually is present in the urine, and the increased serum icterus index, generally is increased. The sedimentation rate tends to be slow in the early stages but becomes faster as recovery occurs. A precipitin test with liver and spleen extracts has been used. Leukopenia may occur. Twenty-five per cent have false posi-

tive serologic tests for syphilis. The urinary coproporphyrin is increased. The bromsulphalein is positive on the third day, the thymol turbidity and cephalin-cholesterol on the fifth day.[33] The barium strip urinary bilirubin test is positive in the preicteric stage. Thrombocytopenic purpura may occur. The prothrombin time may be prolonged.

Course.—The acute signs usually last four to six weeks, but convalescence may be slow, with fatigue, emotional instability and gastrointestinal disturbances, malnutrition and fat intolerance (four to six months). The fever and jaundice may recur (1.5 per cent), sometimes precipitated by exercise and alcohol. Second attacks usually are worse than the first. The severity of the disease, generally, is directly proportional to the age, being relatively mild in nonjaundiced infants and more severe in jaundiced older children and adults.[21] The course in children is shorter than in adults, and the symptoms are milder.

Treatment.—The following remedies have been used with little or no evidence of their value: high protein and carbohydrate and low fat diet, human serum albumin, amino acids, dl-cysteine, fruit juice, gamma globulin, crude liver extract and B complex (IM), penicillin, Aureomycin, Chloromycetin, ACTH, cortisone, avoidance of exercise and alcohol, perfusion of the biliary passages.[21, 34]

Prognosis.—Both epidemic and serum hepatitis usually are benign but may be fulminating and quickly fatal, with or without yellow atrophy, or progress to death in two to nine months without remission.[35] The fatality rate among children is less than 1 per cent[36] but the average in adults is said to be 5 per cent, though it probably is much lower if the mild, nonicteric, and usually undiagnosed patients are included. Among the survivors, the usual duration is four months (six to eight months under 3 years of age),[36] but may last twelve months. A follow-up study of veterans indicated that most residuals were short-term but that a few patients had long-term hepatitis and its residuals (four to six years), either continuously or in multiple attacks. Time encourages healing and the hepatitis is not progressive. Infrequently, acute hepatitis may cause cirrhosis and a fatal hemorrhagic syndrome.[37] Though there were more abnormal liver tests among the veterans who had had epidemic hepatitis than in those who had had the serum variety, the incidence of functional or structural abnormalities of the liver was no greater in patients who had had viral hepatitis than in controls who had had no history or evidence of the disease.[38] Necrosis and yellow atrophy of the liver, nephritis, and pyuria sometimes occur.[39] Infectious or serum hepatitis in pregnancy may produce hepatic necrosis in the fetus, but there is no evidence that it causes anomalies.[40]

Prevention.—For epidemic hepatitis, isolation with stool precautions, as in typhoid fever, for twenty-one days is necessary, and giving gamma globulin (0.01 to 0.1 c.c. per pound) to the family and troop contacts is advisable.[14] To prevent serum hepatitis, transfusions of blood and plasma, especially of pooled plasma, should be avoided, whenever possible, and used only as a last resort. "Fearful that infectious hepatitis (jaundice) is being spread in all civilized countries by virus in blood and plasma, the World Health Organization issued a global

warning; blood donors should be more carefully screened for jaundice carriers, and doctors should give transfusions only when absolutely necessary."[20] Transfusions in surgery, except those absolutely necessary, are more dangerous than many of the operations. From the medicolegal aspect, operative permits should include permission for blood and/or plasma transfusions. Practically all of the microcytic anemias of children can be cured by simple iron sulfate, instead of more expensive mixtures.[41] Those rare children who have reactions to oral iron sulfate can be treated with intravenous iron, and thus avoid the risk of a transfusion which may produce hepatitis. The only pediatric transfusions which are essential are those for hypoplastic and aplastic anemia, leukemia, and severe hemorrhage, and the exchange transfusions in erythroblastosis fetalis. If possible, all of the blood for these patients should be obtained from one donor because if taken from two or three donors, the risk of hepatitis is doubled or trebled.[41] All syringes, needles, blood lancets, etc., must be autoclaved; boiling for fifteen minutes may be ineffective.[11] Gamma globulin should be used for the prevention of measles, etc., instead of blood, plasma, or serum, which, even if ultraviolated,[42] can transmit hepatitis, a worse danger than measles.

CONCLUSION

Hepatitis, both epidemic and serum, is one of the most serious pediatric, hospital, and troop problems, and should be prevented.[43]

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Physiologic Aspects of Pediatrics

ADRENAL STEROIDS IN MENINGOCOCCEMIA

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NUMEROUS reports of recovery from the Waterhouse-Friderichsen syndrome have appeared in recent years. Adrenal steroid therapy has been used in these together with antibacterial agents. While steroids are unnecessary for the successful treatment of most patients with meningococcal infections, the question arises whether they should be administered in an effort to avert the occasional appearance of fulminating meningococcemia rather than to wait for a definite indication. This paper attempts to define the proper indications for their use.

HISTORICAL REVIEW

Several reports[1] in the late nineteenth century described a severe illness usually occurring in infants and characterized by an abrupt onset, a profuse hemorrhagic rash, profound shock, a rapidly fatal outcome, and autopsy findings of extensive adrenal hemorrhages. Little[2] in 1901 collected eleven such cases and felt the etiology was infectious, probably due to the Streptococcus. Waterhouse[1] contributed an additional case report in 1911 and thought smallpox was the most likely cause; MacLagan and Cooke[3] were the first definitely to incriminate the meningococcus in 1916. Friderichsen's[4] report of 1918 was largely a review of the literature; neither Waterhouse nor Friderichsen made any notable original contribution to the syndrome which now bears their name. Subsequently numerous other organisms were found to be occasionally responsible for this syndrome, but at present it is believed that the meningococcus is by far the most common.

No recoveries from the Waterhouse-Friderichsen syndrome were reported from the time of Little's description in 1901 until 1940,[5, 6] when two almost simultaneous innovations in therapy occurred: (1) the introduction of sulfonamides and (2) the use of adrenal cortical extracts, first recommended by Aegerter[7] in 1936. The numerous recoveries that have been reported in the past fifteen years show that the prognosis has radically altered. However, there is no sharp dividing line between uncomplicated meningococcemia and the fulminating form of the disease, and in cases with recovery, adrenal hemorrhages cannot be demonstrated. Confusion has therefore arisen over the criteria necessary for a clinical diagnosis. A clinical term, "fulminating meningococcemia," should be used for those patients with meningococcal infection showing signs of shock or unusually severe hemorrhagic lesions, and the term "Waterhouse-Friderichsen syndrome" for those fatal cases demonstrating both the clinical syndrome and adrenal hemorrhages at autopsy.

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VARIATIONS IN ADRENAL PATHOLOGY

In recent years numerous fatal cases have been described in which the clinical picture was compatible with a diagnosis of Waterhouse-Friderichsen syndrome but in which adrenal hemorrhage at autopsy was either insignificant or entirely lacking. Rich[8] has described a "tubular degeneration" of the adrenal cortical cells in such cases which he believes precedes a hemorrhage. Attempts to reproduce these lesions in experimental animals have been unsuccessful.[9] Ferguson and Chapman[10] subsequently described several fatal cases in which neither adrenal hemorrhage nor tubular degeneration was prominent, though the clinical course was typical of fulminating meningococcemia. They found small thrombi on the capillary and adrenal vein walls, and thought these vascular changes preceded occlusion, stasis, and hemorrhage. These conflicting findings indicate that the mechanism of adrenal hemorrhage and necrosis is unknown. Experimentally, pantothenate deficiency[11] will produce cortical necrosis in rats, and Ingle[12] has reported similar changes following ACTH stimulation in unilaterally adrenalectomized rats. The latter observation suggests that the treatment of fulminating meningococcemia with ACTH, which has occasionally been reported, is inadvisable.

ADRENAL INSUFFICIENCY

The absence of adrenal lesions in some patients dying with fulminating meningococcemia raises doubts as to the role of adrenal insufficiency in the clinical picture. Considerable evidence has accumulated on this point. All reports of surviving patients known to the writer mention the administration of cortical steroids; this uniformity is impressive, even though controls are lacking. Laboratory evidence of adrenal insufficiency has also been sought; the few available studies of electrolytes and plasma volume[13-15] have occasionally but not usually revealed changes characteristic of the addisonian syndrome; there may have been insufficient time for these effects to appear, since total adrenalectomy in animals is compatible with survival for much longer periods than the forty-eight hours within which fulminating meningococcemia usually runs its course. Ebert and Stead[16] studied shock in severe infections caused by organisms other than the meningococcus, and they found no diminution of either plasma volume or venous return. These findings contrast with the picture characteristic of both surgical shock and adrenal insufficiency. Evidence favoring the concept of adrenal failure has been found by other investigators,[17, 18] who observed that the circulating eosinophil count failed to fall in fulminating meningococcemia and cite this as evidence of adrenal insufficiency. Kelley[19] also recently reported a single instance in which blood corticoids of such a patient were found to be only one-sixth of the normal value. Hypoglycemia is commonly found.[20] The evidence, as a whole, favors the concept of adrenal insufficiency, though the limiting factor for survival with the severe infection appears to be not the electrolyte-regulating component, but some other function concerned with the organism's ability to withstand the infection.

THERAPY VS. PROPHYLAXIS WITH THE CORTICOSTEROIDS

The cortical steroids are now used by many therapeutically in fulminat-

ing meningococcemia and prophylactically to prevent appearance of this syndrome. Their therapeutic use is not likely to be questioned, since it is based on the observation that surviving patients have uniformly received a combination of antibacterial agents and steroids. The chief point of difference in the management of meningococcal infections now lies in the indications for the prophylactic use of steroids in patients without signs of fulminating meningococcemia. Proponents of a liberal, indiscriminate use of steroids believe that they are harmless and may occasionally avert a fatal outcome. On the other hand, the routine prophylactic use of steroids in meningococcemia could not produce a large change in mortality. Almost all patients developing fulminating meningococcemia have evidence of it when first seen by the physician, and these patients receive steroids for a definite indication. Most other patients never develop the fulminating disease, since it rarely appears after antibacterial therapy has begun. Its incidence after therapy has started is probably below 1 per cent, so that more than 100 patients would be unnecessarily treated for each one who might benefit. Since the effectiveness of prophylactic steroids in averting this syndrome is questionable, such treatment can be recommended only if it is essentially harmless.

UNDESIRABLE SIDE EFFECTS OF THE CORTICAL STEROIDS

Steroids in high doses for prolonged periods have been used in rheumatic fever and leukemia with only infrequent unmanageable side effects. The problem of enhancement of infection by cortisone is probably not serious in meningococcal infections because this organism is extremely sensitive to sulfonamides and penicillin.[21] On the other hand, the adrenal steroids are notable for their unexpected side effects. They or ACTH have precipitated convulsions,[22] psychoses, gastric and intestinal ulcers,[23] and sickle-cell crises,[24] and activated latent tuberculosis[25] and malaria[24] in human beings. They promote the growth in hamsters of heterologous transplants of malignant tissue[26] and, in rabbits, cause a severe hemorrhagic diathesis following administration of endotoxins,[27] such as is made by the meningococcus. For these reasons their routine use in a situation where under 1 per cent of patients may benefit seems to the writer unjustified. They should be withheld in meningococcemia until definite indications appear.

INDICATIONS AND SUGGESTED REGIMEN FOR THERAPY

The two valid indications for the use of steroids are: (1) unusually *severe hemorrhagic lesions* and (2) signs of *shock* (low blood pressure, pallor, cyanosis). Under these circumstances, treatment should be prompt and vigorous. When the patient is critically ill, penicillin and a sulfonamide, intravenous fluids, and corticosteroids should be given immediately. The steroid of choice is hydrocortisone (compound F),[28] and, next, cortisone (compound E). Aqueous adrenal extracts are less suitable because they contain the biological equivalent of only about 0.1 mg. cortisone per cubic centimeter, but if the pure steroids are not immediately available the extracts may be used initially. Desoxycorticosterone is of doubtful value. No definite dosage schedule for hydrocortisone or cortisone has evolved, but the tendency has been toward larger doses. A large dose, suitable for a severely ill patient during the first six hours

of therapy is 4 mg. per kilogram per six hours for an infant, 2½ mg. per kilogram per six hours for a child, and 1½ mg. per kilogram per six hours for an adult. These doses are roughly equivalent by surface area. At least half of the dose should be given by continuous intravenous infusion over the six hours and the remainder intravenously at the earliest possible moment. Only compound F is available for intravenous use. If compound E is used initially, it must be given intramuscularly and is therefore less desirable, since speed is critical. Dosage during succeeding periods can be revised as the clinical condition warrants. Hodes and associates[18] have suggested that steroid dosage should be at least sufficient to keep the blood eosinophil count at or near zero. The initial high dosage may have to be maintained for twenty-four to forty-eight hours, but following the critical period, steroids should be given in decreasing doses either orally or intramuscularly over the next two or three days. ACTH gel[29] in doses of 4 mg. per kilogram per day for an infant, 3 mg. per kilogram per day for a child, or 2 mg. per kilogram per day for an adult should be given during the last two days of steroid administration to alleviate the transient period of adrenal suppression which follows steroid administration. During the initial critical period, transfusions and intravenous fluids are essential, but with high steroid dosage, excessive administration may present a problem. Sodium, in particular, should be kept at the minimum figure estimated for rehydration (if this is necessary) plus 1 or 2 meq. per kilograms per day for maintenance. Heart failure and cardiac arythmias have been reported,[18, 29] with excessive fluid retention from therapy as a possible factor; but the frequent occurrence of toxic myocarditis from the infection itself makes interpretation difficult. Digitalis should be given if failure occurs. Corticosteroids lower the convulsive threshold, and convulsions following their use in fulminating meningococcemia are reported.[29] These may have been due to the meningitis, but unnecessarily prolonged therapy is to be avoided.

Recently, norepinephrine has been used together with antibacterial agents and adrenal steroids. There has been little experience with this regimen, but some experimental[30] and clinical[31, 32] evidence supports its use. A suggested regimen[32] of therapy with norepinephrine is to add 4.0 mg. (4 c.c. of 1:1,000 solution) to each liter of intravenous fluids and adjust the rate of administration to an amount which will maintain blood pressure above shock level. Further trials are warranted.

Rapid treatment has first priority, but no legitimate opportunity should be lost to obtain and report data much needed for intelligent care of these patients. Serial observations on blood pressure, venous pressure, serum Na and K, eosinophil counts, blood volume, hematocrit, electrocardiograms, and electroencephalograms would be valuable, particularly in their relationship to steroid therapy and the clinical course of the patient.

SUMMARY

The history of the Waterhouse-Friderichsen syndrome is reviewed and the indications for therapy with adrenal steroids evaluated. Patients with meningococcemia should receive adrenal steroids if they develop unusually severe hemorrhages or signs of shock. A regimen of therapy is outlined.

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Psychologic Aspects of Pediatrics

DISTURBED SEXUAL BEHAVIOR IN CHILDREN AND ADOLESCENTS

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THE etiological factors in disturbed sex behavior among children and adolescents have been studied by Rabinovitch, a member of the Michigan Governor's Study Commission on the Deviated Sex Offender.[1] He found that schizophrenia and mental retardation are factors in only a small proportion of cases. The group referred to as psychopaths, and consisting of children reared in an affectionless and generally nonsocial environment, also contribute only a small proportion of offenders.

SOCIAL CAUSES

In a sizable number of cases the etiology is social rather than psychiatric. When children, otherwise normal, are transplanted to new areas where the social taboos are different from the old, they may find themselves transgressing the law without being aware of it. Other social factors mentioned by Rabinovitch are the loss of the force of moral principles and the thrill of a new experience.

PSYCHIATRIC CAUSES

Experiences or influences directly bearing on sexual behavior are at the root of many sexual problems in children and adolescents. A history of early genital experience with adults is one of the commonest findings. Such experiences generate an urgent need for continued gratification at a genital level. In most of these cases, genital contact had taken place many times over an extended period; a single experience does not seem to be sufficient to establish a compulsive need for repetition. It is possible to relieve such youngsters of their tensions through treatment in an institution.

Another fairly frequent cause of disturbed sexual behavior in children and adolescents is an unconscious seductive attitude on the part of a parent. Rabinovitch relates the case of a family in which the father died when the patient, a boy, was 6 years old. The mother had the youngster sleep with her; she bathed him and stimulated him in subtle ways without being aware of the effect her behavior was having. In this way tensions were built up in the boy which found their expression in attempted rape at the age of 14.

According to Johnson,[2] parents may unconsciously encourage delinquency in their children in order to achieve a kind of vicarious satisfaction. Rabinovitch has found that this mech-

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anism is operative in a number of sexually disturbed children. It is more frequent in girls than in boys.

Probably more common than parental seduction or suggestion are overly restrictive and inhibiting attitudes. Children brought up in such an atmosphere feel guilty and apprehensive about their normal sexual preoccupations. Overt symptoms, when present, are generally expressed in somatic complaints, school difficulties, or withdrawal, but, occasionally, when the home has been unduly inhibiting, impulsive sexual aggression may result.

In many cases Rabinovitch relates the disturbance to home situations which are psychologically unsatisfactory in a number of different ways. One common factor in the homes is a lack of opportunity for the development of feelings of comfort, ease, and pride in identifications. When parental relationships are unsatisfactory and the emotional climate in the home is a disturbed one, the child, lacking a proper model which he can imitate, may find difficulty in establishing clarity as to his sex role. In these cases disturbances are more likely to be manifested by inner conflicts and by difficulty in normal sex functioning rather than by overt sexual misbehavior.

An important consideration in some reports of sexual assault is the motivation of the plantiff. In certain instances the complaint stems from the disturbed inner life of the supposed victim rather than from the alleged act. A youngster's unwarranted accusations, arising from his own hysterical reaction to an innocuous incident or from his distorted fantasies, may play havoc with the life of an adult; and, conversely, an adolescent may be falsely accused of sexual delinquency by an emotionally disturbed adult.

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Pediatric Profiles

Alfred Fabian Hess

(1875-1933)

ONE of the outstanding figures in the development of American pediatries was Alfred F. Hess, a scientist and investigator of the first rank, whose contributions helped to spread the fame of American pediatries all over the world. To realize the great variety of the subjects he investigated and the number of his publications, one has but to turn to his *Collected Writings* published in 1936, three years after his death. There in two volumes, each more than 700 pages, are presented almost 200 papers contributed to scientific publications, ranging in subjects from purely clinical observations such as "Carotinemia: a New Clinical Picture," to technical papers such as "The Destructive Effect of Oxidation on Antiscorbutic Vitamin." In addition to these publications, Hess wrote two important books, one on scurvy and one on rickets, in which he presented the fruits of years of careful observations and brilliant discoveries in the fields to which he devoted his life. All of this in spite of the fact that he died in his prime at the age of fifty-eight.

Alfred Fabian Hess was born in New York City in 1875, the son of Selmar and Josephine Hess. He attended school in New York City, went to college at Columbia for a year, then transferred to Harvard where he received his B.A. degree in 1897. Deciding on a medical career, he entered the College of Physicians and Surgeons in New York City, graduating in 1901. After an internship of two and a half years at the Mount Sinai Hospital, he went abroad for two years and continued his training in Prague, Vienna, and Berlin. During this period on a visit to New York, Hess married Miss Sara Straus. Their married life was a very happy one. They were both interested in social welfare, a field in which Mrs. Hess is still most active today. They had four children of whom two daughters survive.

On returning to the United States, Hess worked at the Rockefeller In-

stitute for a year and a half but then decided to go into practice. He was fortunate in that both he and his wife came of well-to-do families and although he was much interested in private practice, he did not need it for earning a livelihood and was able to devote the major part of his time to research. Although his practice was never a very large one, he inspired great confidence in his patients and spent much time with each case. He was an excellent observer and diagnostician. I recall a baby running a high fever that he was asked to see in consultation. He was told that the physical examination was negative, but as soon as he saw the child he called attention to two tiny papules on the buttocks, which he thought might be tuberculides. The baby proved to have miliary tuberculosis.

Much of Hess's early scientific work was done in the laboratories of the New York City Department of Health where he was associated with Dr. William H. Park. Later in his career he transferred his laboratory to Columbia University. He was always contented to work in modest surroundings, and Mrs. Hess tells an amusing story of a young Japanese student who had been sent from abroad to work with him. When this young man found that Hess had only four rooms assigned to him, he refused to work there because "this was not an Institute."

Hess's clinical material was drawn from the wards of the Beth Israel Hospital in New York City, where he headed the pediatric service, and from the Home for Hebrew Infants. The latter was his pet and, together with Dr. Sidney Haas, he transformed it from a very poorly equipped institution into a model of what such a home should be. He introduced the very latest type of equipment and with excellent personnel made it a real show place. Many of the infants there remained as inmates for years, thus making the clinical material especially valuable for long-term nutritional research needed in a study of chronic diseases such as scurvy or rickets. It was there that, as adjunct visiting physician, I first became acquainted with Dr. Hess. To make rounds with him was a real treat. His remarkable powers of observation and deduction and his thoroughness were qualities that impressed me most. He was always ready to discuss cases and one would be sure to come away with new ideas after talking with him.

On looking back now, I realize how far in advance of his times he was in medical matters over forty years ago. He urged the nurses to "mother" the babies, to pick them up and play with them; he had the walls of the wards painted in colors rather than white, to overcome the monotony; he had victrolas installed —all this to make for happier babies. Another instance of his prophetic type of mind was when he mentioned to me that while people were warned against the danger of eating food cooked in copper vessels, he himself was of the opinion that copper might be useful and even necessary as an adjunct to iron in preventing anemia. I remember one day meeting Hess on the ward when he pulled out of his pocket a small bottle containing little, white kernels, which he shook and looked at reflectively, saying with an almost reverent voice, "These are

wheat germs—they are going to turn out to contain wonderful substances." This was in 1912, the year that Casimir Funk coined the word "vitamin."

Another example of Hess's careful observation and clever deduction is revealed in the following note published by him in the *Journal of the American Medical Association* in 1908. It is, I think, worth quoting in full:

Car Conductors as Disseminators of Tuberculosis

When riding in the surface cars most of us, no doubt, have inwardly rebelled against touching the transfer tickets handed us by a conductor, who either has just held the entire pad of tickets between his teeth or has smeared the proffered one with his freshly-licked fingers. For years I also had regarded this merely as a disgusting trick, but when about two months ago a conductor of markedly phthisical habitus handed me a transfer ticket treated in his manner I thought it worth while to find out whether this nasty practice might not likewise be a dangerous one. Accordingly I at once made a smear from the ticket on a slide which I had with me. On staining this specimen little was to be seen. However, a few mornings later I rode in a car with the same conductor, who, as I now noticed, coughed slightly. The car was an open one, so that he used his hands for support and held his pad in his mouth. A smear made from a ticket this time, when stained with carbol fuchsin and decolorized, showed a few definite tubercle bacilli.

Comment on this condition of affairs seems unnecessary. The deduction is simple. This man, day after day, was distributing tubercle bacilli broadcast among passengers. The remedies for this evil which suggest themselves to me are: (a) Medical supervision of the conductors. There is some such provision on the subway and elevated roads but not on the surface lines. (b) A rule forbidding the conductors to contaminate the tickets with their saliva. (c) The employment by each conductor of a chain by which to hang his pad.

Hess was an indefatigable worker and a voracious reader. When one surveys the enormous amount of careful work he produced, one is not surprised to learn that he had no time for hobbies. He was a serious man and appeared reserved and withdrawn when one first met him. However, on longer acquaintance, one realized that beneath this rather solemn exterior there existed a very kind and warm nature. Later in life he enjoyed telling that as a boy in school he was considered not very bright because he asked so many questions. However, he soon found that his classmates, afraid to ask questions, did not understand what was being discussed. He often said that he learned more by asking foolish questions than any other way. He was endowed with a sort of intellectual aloofness, keeping many thoughts to himself, which trait perhaps helps to explain that at the Rockefeller Institute he was advised to go into private practice as he was not cut out for research work! His staff was not afraid of him but admired and respected him and would go to any lengths to help him in his researches. His method of attacking a problem was carefully worked out, and, after his death, was described at length by one of his co-workers, Mrs. Helen Rivkin Benjamin.* She writes:

Probably the characteristic thing about Dr. Hess's mode of work was that he had a conscious intellectual

*Mrs. Helen Rivkin Benjamin: Research Principles of Alfred F. Hess, 1934, typescript on file at the New York Academy of Medicine.

method which he had developed over a period of years and which he applied, step by step, as one would use a routine technic, to the solution of each problem as it arose in the course of his research. . . . The first step, in this method, was to make sure that he could define in words, clearly and precisely, the problem itself. He next considered, and often listed, the facts which were known on the subject and he thought over each in turn to make sure that it was proved and not merely an assumption. . . . The third step in this method of research was to list the facts which, if he knew them, would solve the problem. Only when the situation was thus sharply defined would he translate it into terms of experiment.

Mrs. Benjamin points out that Hess would say, "Logic alone is not enough. You can't think these things through—you have to try them out." Experiments had to be simple. The biological methods were the ones he preferred. He was not interested in complicated methods nor in tricky apparatus. He was not deft with his fingers and did not enjoy building or designing apparatus. As Abraham Flexner* points out, in spite of the fact that many of Hess's problems dealt with various basic sciences, such as chemistry, physics, and bacteriology, he himself was master of none of these and was always ready to turn the problems over to the proper authorities for solution. To quote Mrs. Benjamin again:

Thus he was probably the first to suggest the use of spectral absorption bands in the identification of sterols, though he himself was not familiar with this branch of physics.

*I am greatly indebted to Dr. Abraham Flexner for the use of material contained in his excellent biography of Dr. Hess prefacing *Collected Writings of Alfred F. Hess*, published by Charles C Thomas, 1936.

Again, when the problem of irradiated foods led him into steroid chemistry, he turned to Windaus to carry on the work. In this case as in many others it was necessary to urge that the problem be pursued. The outcome was not only successful from the standpoint of the advancement of knowledge, but was important in causing a change in the application of organic chemistry itself. Before that time few organic chemists were interested in biological questions. Since then there has been a sharp change and their interest has been drawn more and more in that direction.

He carried a small card in his pocket and wherever he went he could be seen pulling out the card and jotting down some idea which had just come to him of something to be done at the laboratory, at the Home for Hebrew Infants, or at his office. (The card had a neat column for each of these three places.) He often said that his whole life was a race with that card, but he never caught up.

Dr. Edwards Park tells me that Hess made a practice of spending an hour every evening alone, quietly going over in his mind the problems and experiments in which he was engaged.

As was mentioned before, the variety of the fields to which Hess made valuable contributions is remarkable. He enriched our knowledge of blood diseases by studies on blood coagulation, purpura, the capillary fragility test, and by the discovery of a tissue extract (thromboplastin) widely used as a hemostatic (1915). In 1912 he introduced the use of the duodenal catheter in infants and by its means investigated

the physiology and the pathology of the infantile digestive tract. He was the first to use convalescent serum to terminate an epidemic of mumps. He first described the clinical picture of carotinemia. Dr. Edwards Park who helped edit Hess's papers for the *Collected Writings* summarizes his achievements in part as follows:

Hess was the first in this country to realize that no provision had been made for the infant in the tuberculosis program and his papers and agitation resulted in the founding of the first sanatorium for the infant already infected with tuberculosis or liable to be exposed to tuberculosis in this country. Hess reached the conclusion that the bleeding in scurvy, which is so characteristic a feature of that disease, is due to changes in the structure of the capillaries and not to changes in the composition of the blood. This view that in scurvy the capillaries become diseased so that they leak has been upheld by the work of Aschoff, Wolbach and others and is the view generally accepted at the present time. He was the first to suggest the removal of the spleen as a therapeutic measure in purpura. Hess was the first to show that vitamin C, the absence of which in the food causes scurvy to develop, is destroyed especially easily by oxidation. . . . The discovery that oxidation was the great enemy of vitamin C revealed simple ways for preservation of the vitamin in the cooking and canning of fruits and vegetables and in the preparation of condensed and dried milk. The simple ways consisted in methods of treatment which avoided the possibility of oxidation. Hess demonstrated that canned tomatoes retained their vitamin C almost completely. This discovery, though seemingly of trivial importance, had great practical consequences, because it at once made various canned foods available the year round as dependable and cheap sources of vitamin C and, incidentally, revolutionized the process of canning fruits and vegetables which contained the vitamin. . . . At a time when the foremost students of rickets the world over were almost unanimous in teaching that cod liver oil was useless in rickets and that its reputation as a specific was legacy from the inaccurate observations of the past, Hess saw clearly, as a result of his own carefully controlled clinical observations, that the oil actually possessed specific curative properties in that disease. A few years later Hess's observations were corroborated by investigators in many parts of the world through the use of the X-ray and chemical measurements of the calcium and phosphorus in the blood. Their studies showed that few drugs possess so specific curative properties in any disease as cod liver oil possesses in rickets. The discoveries of the principle of the creatability of vitamin D in food substances through irradiation with ultraviolet light was Hess's greatest achievement and ranks as one of the great discoveries in medicine. Hess was the pioneer in the development of "irradiated" milk and first developed "yeast" milk. He showed that irradiated milk is more effective in the cure of rickets in infants, as measured in terms of rat units, than milk or viosterol, and at the time of his death was engaged in elucidating the reason. His isolation of the vitamin D substance in the sterol fraction of the food was also a notable accomplishment. Hess was the first to use the spectrographic method for the study of vitamin D. This method was extensively used by scientists for the isolation of vitamin D and is one of the main reliances at the present time in the study of the vitamin. Hess developed an important method for the biological assay of vitamin D in the blood and excreta. Finally he showed that units of vitamin D vary in their effectiveness in infants according to the kind of antirachitic agent employed.

One of the most interesting features of Hess's work is that in spite of his great ability as a scientist and laboratory worker, he managed to remain a pediatrician in the true sense of the word. He was not only interested in his laboratory work as such, but always kept in mind the application of his results to furthering the welfare of the child. This, I think, accounts for the fact that even while working on pure laboratory research, he was at the same time able to deal with and interest others in the social aspects of pediatrics and in the public health problems of childhood. His interest in tuberculosis led to work on the purity of cow's milk and the value of pasteurization, not only of milk but of butter. He founded the Preventorium, an institution that cared for infants suspected of early tuberculous infection or exposed to infection. The Preventorium, located at Farmingdale, New Jersey, began with forty patients. At the present time it has 200 beds and now is also used as a convalescent home for children recovering from tuberculous meningitis. Dr. Hess's widow is still active on the board of directors.

Hess's accomplishments brought him many awards and academic honors. He delivered a Harvey Lecture in New York, the Cutter Lectures at Harvard, the Ingleby Lectures at Birmingham, England. He was given the degree of Doctor of Science by the University of Michigan. In 1927 he received the John Scott medal given by the Franklin Society, and four years later the John Mather Smith award for his work on nutrition, especially scurvy and rickets.

Hess's greatest ambition was to be the recipient of a Nobel Prize, for he felt that his studies on the steroids and radiation in relation to rickets were of greater importance than any other work he would ever produce. That he did not receive this award was a great disappointment to him and to many of his friends and associates who still think it should have been his. In his work on the antirachitic vitamin he became convinced that it was intimately connected with the irradiation of a steroid compound. Not being sufficiently well trained in chemistry, he proposed the problem to a number of American chemists, who showed little interest in it. Hearing that a German scientist, A. Windaus, had special knowledge of steroid chemistry, he wrote to him imploring him to take up the problem. Windaus also at first was not interested, but Hess was not to be discouraged and kept on urging by letter and by cable until Windaus finally capitulated and decided to take on the problem, which in a relatively short time he solved, discovering that ergosterol was the particular compound involved, and that when this provitamin was irradiated, the antirachitic vitamin was produced. That was in 1927. "The following year Huldschinsky, Steenbock, Hess and Windaus were proposed for the Nobel Prize. The examiner came to the conclusion that while the discovery of the antirachitic vitamin was the greatest advance in vitamin research since its beginning, the credit was shared by so many scientists that there were insurmountable obstacles to an award. The same year, however, Windaus received the Nobel Prize in chemistry 'for his researches

into the constitution of the sterols and their connection with the vitamins.' ''* In his speech of acceptance, Windaus gave Hess credit for his work in the field and later was instrumental in obtaining for Hess an annual subsidy for his researches from the German chemical firm, I. G. Farben Gesellschaft.

Many academic positions in various American universities were offered to Hess, but he declined them all. These were wise decisions on his part, for he realized that a professorship entailed much time spent in the detail work of organizing and running a large teaching department. To give sufficient time to the duties would have left him so much less for research and would have distracted his mind from what was his true life-work.

Hess kept on working under high pressure although he had been warned by his physician because of hypertension and mild cardiac attacks that he must reduce his pace and must give up all public speaking. He disregarded this advice, and on December 5, 1933, insisted on speaking at a nurses' graduation exercises. On his way home with a colleague he suddenly collapsed and died in his automobile.

*Quoted by permission from *Nobel, the Man and His Prizes*, edited by the Nobel Foundation, published by the University of Oklahoma Press, 1950.

In his early death (he was only 58 years old) pediatrics lost an investigator who was regarded both here and abroad as one of the outstanding scientists of his time. On the morning of the day on which he died, he telephoned to his wife who was out of town, to tell her that he had finally solved a very important problem on which he had been working. He had the habit of turning over and over in his mind the results of his experiments and not putting them down on paper until he had come to some definite conclusions and was convinced that they were correct. As a result, nothing in the way of a solution was found in his papers.

Most of the younger men in pediatrics do not know very much about Hess. Some of them know his name but few know his achievements. However, neither a bust, a portrait, or even a biography necessarily keeps the memory of a man green. He is best remembered by his accomplishments, and though the pediatrician of today may not realize it, when he diagnoses latent scurvy, when he avails himself of irradiated foods or vitamin D, when he recognizes carotinemia, or uses a hemostatic such as thromboplastin, he is truly remembering that extraordinary pediatrician Alfred Fabian Hess.

Murray H. Bass

Comments on Current Literature

ENTERIC VIRUSES

THE smaller viruses are true intracellular parasites requiring a living cell in which to grow and multiply. Their presence can now be detected in a number of ways, but of these the tissue culture method, practical and relatively inexpensive, has permitted extensive investigations which hitherto would have been considered onerous, unwieldy, and prohibitively expensive. The effective use of antibiotics, notably penicillin and streptomycin, has proved of inestimable value in rendering contaminated specimens free of bacteria, supressing detrimental bacterial activity, and permitting detection of the slower effect of viral agents on the living cell.

Recently Ramos-Alvarez and Sabin[1] have made good use of the roller-tube tissue culture method, employing cynomologus monkey kidney cells, in a study of the enteric viral flora of 1,566 healthy children. Rectal swabs from each child were collected, in July and August of 1953, from children of lower income families living in Cincinnati and Xenia, Ohio. After appropriate preparation, each rectal specimen was inoculated on monkey kidney in tissue culture. When evidence of cytopathogenic effect appeared, subculture passages were carried out, and subsequently identification of the newly isolated agent was undertaken.

From the 1,566 specimens, thirty-one cytopathogenic agents were recovered. Five of these agents proved to be strains of poliomyelitis virus, three of Type II (Lansing) and two of Type III (Leon); one strain of virus isolated was identified as Coxsackie Group B; and twenty-five agents producing definite cytopathogenic effects were recovered which were not immediately identifiable. It is of interest that the five strains of poliomyelitis virus isolated from healthy children during this nonepidemic period were restricted to Types II and III. During epidemic periods, Type I (Brunhilde) has been recovered with the greatest frequency, both from patients and from healthy contacts. The authors have in progress at the present time a further study of rectal swab specimens from about 1,800 young Mexican children in an attempt to ascertain "whether Type I poliomyelitis viruses may be encountered with greater frequency in a population which is presumed to have a higher rate of inapparent or unrecognized infection with all types of poliomyelitis viruses."[1]

These investigations by Ramos-Alvarez and Sabin are interesting in the light of earlier studies by Melnick and Ågren[2] in Egypt, and by Gear and his associates[3] in Africa, which indicated the presence of virus strains in stool specimens of normal infants: poliomyelitis, Coxsackie viruses, and several unidentifiable agents. While from time to time the paralytic attack rate for poliomyelitis in these particular areas increases appreciably, the disease has not occurred apparently in widespread epidemic form. It has been suggested that infection of the population takes place early in life, conferring immunity by natural means.

Antibody studies in normal children likewise are of interest in this

connection. During an epidemic of poliomyelitis in 1948 in Winston-Salem, N. C., Melnick and Ledinko[4, 5] investigated antibody levels to all three types of polio virus in a normal young population living in the area through the epidemic period. The results showed that there had been an increase in the incidence of antibodies to Type I poliomyelitis virus in all age groups, the increase being most marked in children under 4 years of age. Postepidemic specimens indicated that children of all age groups had increased levels of antibody to Lansing virus (Type II), except those who were under the age of one year. In the case of the Leon virus (Type III), there was no appreciable change in the level of antibody during this period; in all age groups, with the exception of newborn infants, who showed a rise in titer early in life, the findings for Type III antibody were essentially the same in serum specimens collected early in the epidemic or before the epidemic, and late in, or following, the epidemic.

All five strains of poliomyelitis virus isolated by Ramos-Alvarez and Sabin[1] were established and identified (Types II and III) in tissue culture of monkey kidney. Kidney culture fluid was inoculated intracerebrally and intraspinally into cynomolgus monkeys, and the results obtained in each case "conformed to the pattern of intracerebrally avirulent, spinal paralytogenic variant viruses" described previously by Sabin and his associates.[5] The Type II poliomyelitis viruses were pathogenic for mice by the spinal but not by the intracerebral route, and the Type III viruses were avirulent for mice by both routes.

The other cytopathogenic agents (twenty-five strains) isolated from rectal-swab specimens would appear to constitute a new group of enteric viruses, and have been designated by the authors as the HE (human enteric) viruses. None of these new enteric viruses was related antigenically to the strains of "orphan viruses" recovered by Melnick[6] and by Steigman and his associates[7] from stool specimens obtained from patients with suspected nonparalytic poliomyelitis. Human gamma globulin contains appreciable antibodies to all except one of the newly isolated HE viruses, suggesting that the agents are rather widespread in the human population. Further investigation may reveal the nature of this infection, whether it is subclinical, or associated with recognizable clinical manifestations, etc. Twenty-two of these twenty-five new viruses belonged to three distinct serologic types (eight were of the so-called Type 1, eleven were of Type 2, and three were of Type 3); three of the twenty-five new strains remained unclassified. The HE 1, HE 3, and the unclassified strains were neutralized by human gamma globulin, while the HE 2 virus was not neutralized by any one of the gamma globulin samples employed. The HE 1 and HE 2 strains produced neither paralysis nor any significant lesions after intraspinal inoculation in cynomolgus monkeys.

These strains of virus, five poliomyelitis strains, and a group of enteric viruses, the exact nature of which is not understood as yet, were encountered in a study undertaken by the authors[1] in an attempt to "determine whether or not avirulent poliomyelitis viruses occur in nature." Only healthy children were selected, and those who had had no known contact with any person having clinically recognized poliomyelitis. Such avirulent strains of poliomyelitis virus and other enteric viral agents are of considerable academic interest, and at the same time pose new epidemiologic problems.

RUSSELL J. BLATTNER

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Correspondence

WAITING FOR A PEDIATRICIAN?

WE moved to a metropolitan area five years ago, and since then, because of several short moves to different suburbs, we have become acquainted with a number of pediatricians. We did not change doctors primarily because we were moving to a new locale. Although it is handy to have a doctor's office near by in case of emergencies, had we found what we were looking for, a longer trip to a doctor's office would have been a minor consideration. We changed doctors because we were not satisfied with the services offered. I am convinced that I am not alone in this dilemma.

"I would rather do a hard day's work than take Johnny in for his booster shot. A whole afternoon is just lost, and I come home tired and angry!" This is what one mother said as we sat around comparing notes on the things that happen when we take our children to a pediatrician's office. We were all concerned about the kind of service offered. It was agreed that while we were relieved to know that there is expert advice available when we need it, we wondered why the experience in acquiring it could not be a happier and more pleasant one.

Some mothers have lived in one community for a long time and have a pediatrician they swear by. They do not share these feelings. However, many mothers who have moved more frequently do react this way.

This is an age of specialists. Pediatricians are our children's specialists. For the adequate fees they receive, their services should be of the highest standard. In his busy day the doctor often loses that personal touch which is vital in any relationship between seeker and adviser.

What is it that we mothers feel so keenly? Many will answer at once, "Long periods of waiting in his office." These occur so frequently that they are accepted stoically. Appointments are always given with care. ("We can take you at 2 P.M. but not at 2:15; we are running a close schedule.") On that date you rush to finish up the work, arrive at the office usually a bit before appointment time in the hopes you will get out earlier, and then just sit. At first it is in a crowded and usually hot waiting room. You distract and amuse one or more irritable children until finally your name is called.

With relief, you hurry into a small examining room, one generally on the cool side. You strip your child and wait some more. By this time he is tense and apprehensive. While you try to reassure, he finds bottles, taps, scales, and venetian blinds a distracting game. With wishful thinking you open the door and peer down the hall to see more examining rooms, all filled, and perhaps other anxious faces peeking out at you. Finally, when you are ready to settle for anything to get home, the doctor strides in and gets down to business.

Quite often it is obvious that the doctor is under pressure. He is rushed—he, too, knows the waiting rooms and examining rooms are full. It is satisfying to watch him make a quick and skillful examination, but when he offers his diagnosis, too often he phrases this in medical terms when a simpler explanation would be less confusing. Just as you are about to ask a pertinent question he is interrupted by a telephone call. When he returns, he writes his prescription as he talks.

In one office, I always had to trot down the hall after the doctor while he got his prescription slips at the receptionist's desk. He talked over his shoulder as he walked. Not getting the instructions quite clear, I had to telephone later to verify something I had missed.

There are always other things that you want to take up with your pediatrician. Perhaps you need a little reassurance or advice about a current behavior problem. Special trips are not made to discuss John's sudden night fears, or the baby talk to which Mary has recently reverted. You save these up

for the time when you will be going to see him, since preschool children have a habit of becoming sick fairly regularly. Either you do not have the opportunity to ask these questions at all, or, if you do, you wedge them in quickly and then find yourself in the impossible situation of discussing problems in front of your children.

And so comes the letdown—the long wait (it can last anywhere from forty minutes and up), the brief hurried time with the doctor, a five dollar bill, and a knot in your stomach from frustration. You come away with the feeling that if the doctor showed a little more awareness of the feelings of his patients and their families, the whole business could have been handled differently.

What do we expect from pediatricians? I think we expect a little better planning. Waiting long periods does not help a sick or well child. Wondering what is going to happen next makes him tense and anxious. We would like to be seen by the doctor as close to the appointment time as possible. Other professional people including medical specialists with big practices seem to consider the obligation to keep an appointment mutually binding. Granted a good doctor does build up a larger clientele, but satisfactory service should not suffer because of this. In the last analysis, it should not be how many patients are seen in one day, but how satisfying it has been to both the doctor and those who seek his advice. If a doctor finds that he cannot spend adequate time in his daily interviews, then he should hire an assistant. If he is still pressed, then why not limit the number of patients per day?

Mothers feel their time is valuable too. I know if my husband accompanies me, as he has on occasion, the afternoon can turn into an expensive outing. The company that employs him has calculated that it costs $100 per day to keep a man at work. We have spent an entire afternoon dismally aware that this sort of treatment is somewhat highhanded.

We expect courtesy from professional people. I have heard business executives, social workers, and gas station attendants apologize for keeping a client waiting, but I have yet to hear a pediatrician offer the slightest apology for the inconveniences he may have caused. In fact at times I have had the feeling as they made that first appearance that a small curtsy might be in order.

A doctor should take time to be gracious—to remember names—to try to recall a bit of the background of his regular clients. Some parents have been to a university too, and perhaps if this were known or remembered he might not be so likely to talk down to his bread and butter.

Explaining the situation in language the layman can understand is another courtesy we expect. Some mothers do not understand medical terms. If these are all that are offered, they go home to scout out explanations from well-meaning friends who often confuse and alarm them further.

Once the examination is over, a mother would like to go into the doctor's private office, sit down, and calmly hear explanations and instructions. Not on the run, but in a more relaxed atmosphere. This would be the time to bring up those other problems in privacy while the children are elsewhere, even if the nurse has to amuse them while the mother is occupied. Pediatricians in training are taught to stress the psychiatric approach. More and more, medical journals indicate symptoms which are not physical in basis.

Mothers do not want to monopolize a doctor's time, in the office or on the telephone. Perhaps a special time to take telephone calls at one particular hour of the day might be a solution. There are emergency calls naturally, but if a doctor had a regular time for other calls, needless and disconcerting interruptions would be eliminated.

We do not want to be continually seeking a pediatrician who meets these needs. This sort of shopping around is expensive and disturbing. A first visit to a new doctor is always billed higher. A progression of "first visits" is upsetting to both mother and child.

A visit to our very own pediatrician should mean more than the administering of sulfa, or a shot of penicillin. It should be more than a diagnostic confirmation of what is already suspected. A child grows into adulthood needing strong and secure anchors in relationships to key people in his life—his parents, his school contacts, and, not least, his physician.

L. H.

OXYGEN AND RETROLENTAL FIBROPLASIA

I WANT to congratulate Dr. Lanman for his excellent discussion on the control of oxygen therapy for the prevention of retrolental fibroplasia. As Dr. Lanman states, the New York City Health Department's Premature Transport Service limits oxygen concentration even though the trips seldom exceed forty-five minutes. In addition to this, the New York City Department of Health also issued in April, 1954, the recommendations which were approved by the Pediatric Advisory Committee on March 17, 1954, relating to retrolental fibroplasia as follows:

"Recent studies suggest a possible interrelationship between high concentrations of oxygen and the development of retrolental fibroplasia in premature infants. It has therefore been recommended by the Pediatric Advisory Committee that premature infants receive oxygen only as needed, and then in concentrations not to exceed 40 volumes per cent.

"The Committee emphasizes that inasmuch as the concentration of oxygen cannot be reliably measured in terms of liter flow, determinations of oxygen concentrations should be made by means of oxygen analyzers at periodic intervals." It was disseminated to all maternity and newborn services and to the entire profession.

Premature centers have also been requested to report to the Department of Health any cases of retrolental fibroplasia which may occur on their services. Through the periodic hospital inspections and surveys made by the staff of the Maternity and Newborn Division, a careful checkup is also made of the hospital policy with regard to the administration of oxygen to premature infants and the necessary recommendations are made as indicated. A joint advisory committee of pediatricians, obstetricians, and ophthalmologists was appointed. The subject was reviewed and I stated then that "despite previous recommendations of the Department of Health and the Pediatric Advisory Committee that oxygen concentrations of 40 per cent or less be used in the nursery care of infants, at present many hospitals are still using higher concentrations." It was recommended that:

1. Oxygen concentration be used for premature infants in delivery rooms on obstetrical services below 40 volumes per cent and not continually but only when indicated.
2. Concentrations be checked with oxygen analyzer.
3. An ophthalmologist make periodic ophthalmological examinations in all premature nurseries and on all premature infants.

It may be stated that there has definitely been a reduction in the incidence of retrolental fibroplasia among premature infants born in 1954 as compared with those born in 1953. While complete figures are as yet unavailable, we only know at present of four infants who were reported to the State Commission for the Blind who have been born during 1954. This does not mean, however, that only four cases of retrolental fibroplasia occurred in infants born in 1954 for there is a considerable lapse between date of birth and reporting the blindness, but it definitely shows a considerable drop since there were over sixty cases reported during the year 1953.

HAROLD JACOBZINER, M.D.

News and Notes

The following were certified by the **American Board of Pediatrics** at the examination at Detroit, April 1, 2, and 3, 1955.

Dr. Ellis Wentworth Adams, 517 Wildwood Ave., Jackson, Mich.

Dr. Richard Allen, 113 B Rodman Rd., Aberdeen, Md.

Dr. William Blair Anderson, Children's Hospital of Philadelphia, 1740 Bainbridge St., Phildelphia, Pa.

Dr. Irving William Bailit, 1233 Highland Ave., Needham, Mass.

Dr. Henry Welles Baird, III, 116 Woodside Rd., Haverford, Pa.

Dr. DeWitt C. Baldwin, Jr., Department of Pediatrics, University of Washington, Seattle 5, Wash.

Dr. Donn Richard Barber, 809 Steele, Denver 6, Colo.

Dr. Stanton J. Barron, Jr., 287 Billy Mitchell Ave., Ellsworth AFB, Rapid City, S. D.

Dr. Ralph H. Berry, Jr., 413 N. Fifth St., Reading, Pa.

Dr. J. Edmund Bickel, 1700 Frederica St., Owensboro, Ky.

Dr. Veronica B. Binns, 308 Union Station Bldg., Brownsville, Pa.

Dr. Mary Josephine Blood, 647 N. Hillside, Wichita, Kan.

Dr. Richard Alan Bloomfield, 17911 Scottsdale Blvd., Shaker Heights 22, Ohio.

Dr. Donald Buckey Bond, 110 S. Central Ave., Clayton 5, Mo.

Dr. Gardner Champlin, 1037 Alexander Circle, Pueblo, Colo.

Dr. Donald Brook Cheek, Children's Hospital Research Foundation, Elland Ave. & Bethesda, Cincinnati 29, Ohio.

Dr. William M. Clark, Jr., University of Oregon Medical School, Portland 1, Ore.

Dr. Marvin E. Cooper, 4400 W. Pratt Blvd., Lincolnwood 30, Ill.

Dr. James Brendan Corbett, U. S. Army Hospital, Camp Rucker, Ala.

Dr. John Richard Craig, 17 Prospect St., Waltham, Mass.

Dr. Ray William Croyle, 875 Barnes St., New Kensington, Pa.

Dr. Mauricio Martins da Silva, Department of Pediatrics, University of Minnesota Hospitals, Minneapolis, Minn.

Dr. James Ray Doores, 320 N. Kellogg, Galesburg, Ill.

Dr. Saul Kahn Dopkeen, 58 Commodore Rd., Worcester, Mass.

Dr. Rosario P. Drago, 1743 Bussing Ave., Bronx, New York, N. Y.

Dr. David Patterson Dyer, 550 Morris, S. E., Grand Rapids, Mich.

Dr. Leonard I. Ehrlich, 45 N. Station Plaza, Great Neck, N. Y.

Dr. Kenneth Orson Fishler, 465 E. South Temple, Salt Lake City, Utah.

Dr. Harold W. Ford, 10002 Cedar Ave., Cleveland 6, Ohio.

Dr. Irving R. Fox, 1360 Morris Ave., Union, N. J.

Dr. Esther Friedenthal, 22-18 169th St., Flushing, N. Y.

Dr. William Ries Gabbert, 1316 W. 15th St., Owensboro, Ky.

Dr. Richard R. Galpin, 525 Southfield, Birmingham, Mich.

Dr. Louis F. Garben, Jr., 518 Ridgewood Ave., Glen Ridge, N. J.

Dr. Eleanora Cunningham Gordon, 116 Woodside Rd., Haverford, Pa.

Dr. I. Norman Gould, 136 S. Franklin St., Wilkes-Barre, Pa.

Dr. Roger Clifton Grady, Children's Hospital, Detroit, Mich.

Dr. Charles Richard Hawes, 1021 E. 19th Ave., Denver, Colo.

Dr. Richard G. Hodges, St. Lukes Hospital, 11311 Shaker Blvd., Cleveland, Ohio.

Dr. Robert Louis Hoffman, 627 Third National Bldg., Dayton 2, Ohio.

Dr. Erwin Ping-Shen Huang, Children's Memorial Hospital, 1615 Cedar Ave., Montreal 25, P. Q., Canada.

Dr. Nicholas C. Kamakas, University Club Bldg., 607 N. Grand Ave., St. Louis, Mo.

Dr. Charles Howard Kelsall, 3385 S. Bannock, Englewood, Colo.

Dr. Malcolm John Kelson, 1045 Harvard Rd., Grosse Pointe Park 30, Mich.

Dr. Melvin A. Krugly, 6710 West North Ave., Chicago 35, Ill.

Dr. Ruben Kurnetz, 20473 Strathmoor, Detroit 35, Mich.

Dr. Albert Leonard Lamp, Jr., 212 W. 6th St., Erie, Pa.

Dr. Sanford L. Leikin, 6809 Riggs Rd., Hyattsville, Md.

Dr. Marvin R. McClellan, 3425 Glenmore Ave., Cincinnati 11, Ohio.

Dr. Glen E. McCormick, 1411 Wauwatosa Ave., Wauwatosa, Wis.

Dr. Elizabeth Lewis McGee, Bent Rd., Bowling Green, Media, Pa.

Dr. Derrill Byrne Manley, 1313 N. Second St., Phoenix, Ariz.

Dr. Matthew J. Marano, 1041 S. Orange Ave., Newark, N. J.

Dr. LeRoy W. Matthews, University Hospitals, 2103 Adelbert Rd., Cleveland 6, Ohio.

Dr. Howard A. Mermelstein, 5405 Fifth Ave., Pittsburgh 32, Pa.

Dr. Robert Moore, 1840 B Forest, Fort Meade, Md.

Dr. Richard Stewart Nelson, 451 E. Campbell Ave., Campbell, Calif.

Dr. Robert M. Nicholson, Jr., 413 N. Berkley St., Kalamazoo, Mich.

Dr. Gordon Quentin Olsson, Rapid City Medical Center, Rapid City, S. D.

Dr. Robert L. Parr, 3403 E. 38th St., Indianapolis 18, Ind.

Dr. William H. Peirce, Department of Pediatrics, University Hospital, Ann Arbor, Mich.

Dr. Morton E. Pizer, 2019 Clark Ave., Raleigh, N. C.

Dr. Mildred Jane Polniaszek, 5159 Fullerton Ave., Chicago 39, Ill.

Dr. Charles C. Powel, 170 S. Main St., Harrisonburg, Va.

Dr. Klara Judith Prec, 1607 E. 50th Pl., Chicago 15, Ill.

Dr. William Raffel, 2028 16th St., N. W., Washington 9, D. C.

Dr. Donald L. Rogers, 3426 N. Meridian St., Indianapolis, Ind.

Dr. William E. Segar, Department of Metabolism, AMSCS, Walter Reed Army Hospital, Washington 12, D. C.

Dr. Nerissa P. Singh, 5738 S. Maryland Ave., Chicago 37, Ill.

Dr. Charles Robert Smith, 4032nd USAF Hospital, Carswell AFB, Fort Worth, Texas

Dr. William A. Spencer, 5306 Stuyvesant Lane, Houston 21, Texas.

Dr. Donald Karl Stockdale, Babies' Hospital, 167th St. & Broadway, New York 32, N. Y.

Dr. W. William Tornow, 44 West St., Worcester 2, Mass.

Dr. Jack V. Turnbull, 22340 Michigan Ave., Dearborn 7, Mich.

Dr. Judson John Van Wyk, Johns Hopkins Hospital, Department of Pediatrics, Baltimore 5, Md.

Dr. Lewis William Wannamaker, Department of Pediatrics, University of Minnesota, Minneapolis, Minn.

Dr. Creed Francis Ward, 1488 E. 105th St., Cleveland 6, Ohio.

Dr. Ralph Josiah Patrick Wedgwood, Department of Pediatrics, Western Reserve University, 2065 Adelbert Rd., Cleveland 6, Ohio.

Dr. Samuel Weinberg, 15 Dogwood Pl., Massapequa, N. Y.

Dr. Howard Jones Williams, Jr., Apt. B-6, 833 North Ave., Macon, Ga.

Dr. Howard Leroy Wilson, Billings Clinic, Billings, Mont.

Dr. Thomas Buerger Wright, 2711 Iroquois Ave., Waukegan, Ill.

The following oral examinations have been scheduled:

New York, N. Y., June 10, 11, 12, 1955.
Chicago, Ill., Oct. 7, 8, 9, 1955.

The **New York State Department of Health** has made available a special grant of Federal C.C. funds to provide a fellowship in rheumatic fever of one year's duration for a physician with a good general or pediatric background who lacks specific training in this field. Priority in selecting the candidate will be given to a physician wishing to make full-time teaching, research, or public health a career and who has an interest in children's rheumatic fever and related programs. The fellow will have the major part of his training at the New York University-Bellevue Medical Center and at Irvington House, a special institution at Irvington-on-Hudson for rheumatic children, affiliated with the New York University-Bellevue Medical Center. The budget for the fellowship is as follows: (1) stipend, $4,200; (2) travel expenses, $800. Interested applicants should write to the Medical Director, Irvington House, Irvington-on-Hudson, New York.

Book Reviews

Standard Values in Nutrition and Metabolism. Edited by Errett C. Albritton, Philadelphia, 1954, W. B. Saunders Co., 380 pages.

An exhaustive reference book prepared by the National Academy of Sciences–National Research Council, and edited by Professor Albritton of George Washington University. The book contains hundreds of statistical tables relating to values in nutrition and metabolism. It contains material relating to nutrients and their utilization by animals and plants, foodstuffs and feedstuffs, O_2 consumption by man and plants, energy exchange, and the like. The book is intended for those working in the field of research.

Deutsche Nachkriegkinder. C. Coerper, W. Hagen, and H. Thomae, Stuttgart, 1954, Georg Thieme Verlag, 544 pages. Price $7.85.

A first report on studies of growth and development being carried on in a number of communities in West Germany on post-war children of school age. The studies have been supported by funds from the Marshall Plan. The first part of the report describes the material and methods of the study, and details the first findings which form the basis for the long-term studies to be made. It presents the social and family background, the physical findings, and the psychologic studies. The text will be of particular interest to clinics and centers where long-term growth studies are being carried on.

Current Therapy 1955. Edited by Howard F. Conn, M.D., Philadelphia, 1955, W. B. Saunders Co., 692 pages. Price $11.00.

The 1955 volume on *Current Therapy* contains therapeutic methods and measures from nearly 300 contributors. The Editor has succeeded in keeping the text abreast of the newer drugs and methods. Like the previous volumes, it is an excellent text for quick reference.

Textbook of Physiology, ed. 17. Edited by John F. Fulton, M.D., Philadelphia, 1955, W. B. Saunders Co., 1275 pages. Price $13.50.

It is interesting to read the Preface by the late W. H. Howell which appeared in the first edition of the text in 1905. The various editions through the years have kept pace with the many scientific developments in physiology and medicine. Like in almost every other branch of medicine, the knowledge has become so vast that physiology finds itself divided into fields of special interest. Thus the present text contains contributions from thirty physiologists under the editorship of Dr. Fulton. There are many changes in the text since the last edition in 1949, and a number of chapters have been completely rewritten. Beyond question, the text will continue to hold its place as one of the standard texts on physiology.

Advances in Pediatrics. Vol. VII. Edited by S. Z. Levine, M.D., Chicago, 1955, Year Book Publishers, 351 pages. Price $8.00.

The seventh volume of *Advances* contains seven reviews. Caffey discusses the cortical defects in growing tubular bones which are a not uncommon accidental finding in roentgenograms. Meredith Campbell contributes an excellent general discussion of the urinary tract in childhood. Gomez and his associates discuss malnutrition in infancy and childhood, which is obviously a more common finding in Mexican children than in children seen in clinics in the United States. Mannheimer of Stockholm presents an interesting chapter on phonocardiography, which is not in common use in the United States, and discusses the findings in various types of cardiac disease. Shwachman and his associates at the Children's Medical Center in Boston discuss mucoviscidosis with emphasis on the importance of studies of the duodenal juice in establishing the diagnosis. Swenson of Boston reviews his surgical experiences with congenital megacolon.

Cardiac Anomalies. Vincent Moragues, M.D., and Chester P. Lynxwiler, M.D., Baltimore, 1954, Williams & Wilkins Co., 92 pages. Price $6.50.

The authors have attempted to present a correlation between the clinical and pathologic findings in congenital heart disease. The congenital defects are grouped in seven main divisions as septal defects, valvular anomalies, and aortic arch anomalies. Under each main heading specific lesions are discussed. Most of the book is taken up by 157 large illustrations. These are excellent, particularly the photographs of the gross pathologic lesions. The clinical discussion consists simply of a listing of the clinical and laboratory findings. From the standpoint of an atlas, it is not as complete as the Abbott atlas, and the clinical discussion is too sketchy. Its chief interest lies in the large-sized illustrations.

Practical Fluid Therapy in Pediatrics. Fontaine S. Hill, M.D., Philadelphia and London, 1954, W. B. Saunders Co., 275 pages. Price $6.00.

The text is preceded by a glossary of terms frequently used in fluid and electrolyte discussions so that the reader can refresh his memory or correct any misconceptions. The body of the book is divided into three parts. In the first section, "Basic Physiological Principles and Their Clinical Significance," water metabolism, fluid, and electrolyte distribution, electrolyte imbalance, and kidney function are discussed. The author then discusses the pathological physiology and treatment in situations such as diarrhea, diabetic acidosis, fluid therapy in renal disease, including descriptions of history and methods of diagnosis. The third section describes the techniques available for drawing blood and administering parenteral fluids.

The author's treatment of the subject matter is straightforward without any reference to the many existing controversies. Repetition and lack of detail detract from the value of the book for some, but medical students, house officers, and busy practitioners should find it of much help in treating clinical problems.

M. R. B.

Health Supervision of Young Children. Prepared by the Committee on Child Health of the American Public Health Association, New York, 1955, American Public Health Association, 179 pages, Price $2.00.

This manual on health supervision was prepared by a committee under the chairmanship of Dr. Samuel M. Wishik. It was reviewed and approved before publication by committees of the American Academy of Pediatrics and the Association of State Maternal and Child Health Directors. It further bears the endorsement of the Children's Bureau and the National League for Nursing. The text, written by Nina Ridenour, is divided into two parts of approximately the same length: I. Health Supervision; II. The Child Health Conference. The first section stresses the changing concepts of medical responsibility for young children. As a part of this, considerable material on parent counseling is presented in keeping with the stress placed on psychologic problems, parent-child relationships, and personality. While this contains some sound advice, some of it, as the ten-page discussion of the doctor and nurse as skilled interviewers, reads like a primer written to the level of children of high-school age. In stressing the "changing concepts," so much of the text is used in this way that the discussion of the physical side of health supervision, a matter of equal or even more importance, is sketchy and incomplete.

The second section on the Health Conference is an excellent discussion of the subject. In an Appendix to this section, details are given as to satisfactory physical equipment, records, immunization procedures, and the like.

In the foreword it is stated that this utilization of space was deliberate, and may seem disproportionate. This is the reaction of the reviewer. It has resulted in an unbalanced discussion of health supervision. This is certainly true for one of its purposes, as "a guide for practicing physicians," if not for the second stated purpose, "and child health conference personnel."

Editor's Column

IN SEARCH OF A PEDIATRICIAN

WE publish under Correspondence in this issue a letter received from an articulate young mother who feels she has not been able to obtain the services she desires from the pediatricians she has encountered. The letter interested the members of the Editorial Board, and the Editor has found their comments of equal interest. Obviously, the individual pediatrician will react to the letter in the light of his own experiences and methods of practice. The Board was in agreement that the letter should be published, with the thought that it may help in arranging our activities to meet the search of mothers such as Mrs. H. for a "satisfactory" pediatrician. It does no harm for us to see ouselves as others see us, even if the seeing eye is somewhat jaundiced. Excerpts from the comments by one member of the Board were as follows:

I think it is worth while to publish the letter—it may make a few practitioners wince, and may make the good lady feel a little more self-righteous than I believe is her just due. Every pediatrician encounters a Mrs. H. from time to time. She appears full of complaints about previous attendants—is completely delighted with the new doctor who must be the final answer, but gets unhappy with him as she has with the preceding ones and departs to search further for someone to her liking.

It is not altogether too bad that Mrs. H. never gets quite as good care as the mother who sees the same pediatrician over the years, becomes acquainted with his personality, his methods, and accepts even his imperfections.

The pediatrician must take the bitter with the sweet and, like any tradesman, must give his best efforts to every patient who enters his consulting room. Most of us learn that there are some patients whom we simply cannot please no matter how hard we try. Some parents will be endlessly grateful for the care of a serious situation in which the pediatrician has been groping amidst his own uncertainties and has been most unhappy about his efforts. Other parents will be extremely bitter over a less serious problem where they have demanded infinite patience, time and effort.

Waiting for a physician is always an annoyance but I do not see how it can be sometimes avoided. Mrs. H. would have the busy pediatrician have numerous assistants, but I am sure she means that these should take care of the *other* patients in the waiting room.

I am constantly saddened by the fact that patients single out diametrically opposite characteristics as the reason for their affectionate regard of a pediatrician: One is admirable because he always bounces in bright and early and another is equally loved because he drags in long past his dinner hour. One never permits any patient to waste his time and conscientiously keeps the flow of patients through his examining rooms so that no one is ever kept waiting—still another is ideal because no matter how many patients are waiting, no matter what the press of time, he is never

too busy to give the anxious mother endless attention for the solution of problems of infinitesimal importance.

Mrs. H. will never get a pediatrician to her liking. She will pursue her petulant way with her anxious child from pediatrician to pediatrician while her husband continues to lose his very valuable time. My heart bleeds for her—but not very much.

Another comments:

Undoubtedly there are pediatricians to whom her criticisms would apply, but there are a great many more—probably the majority—who do not run assembly-line businesses.

While I think Mrs. H. may have a just criticism if she has happened to run into the assembly-line type of pediatrician, there is a possibility that the fault may be entirely hers. I think her letter might do some good by focusing attention on the objections of the public to crowded and hurried office practice. Her comment that pediatricians with such practices should either get an assistant or not make so many appointments seems entirely justified. John Hubbard's survey of child health services and facilities brought out the fact that some pediatricians saw as many as eighty patients a day. I do not see how a pediatrician can see more than twelve or fifteen patients by appointment in an afternoon, and do them justice. It is difficult to escape the conclusion that pediatricians who see from fifty to eighty patients a day in their offices are more interested in finanical returns than in rendering the services of a specialist.

A third made the following comment:

Mrs. H., I judge from her letter, belongs to that difficult type of mother who comes to the office with an appointment for one child. Just as you have finished and her appointment time is about up, she remarks that she did not bring Johnny as he was not sick, but wants to know what to do about his wetting the bed ("a little advice about a current behavior problem," as she states). To answer this question intelligently would take at least a half hour of questioning and explanation. She bitterly resents your brushing off the question and not taking the time from the other waiting patients with appointments.

Obviously she has encountered some pediatricians who are interested in quantity rather than quality of service. Personally, I have never been able to see more than ten or twelve patients in an office session without having the feeling that it was unsatisfactory medical practice, both for the parent and child and for myself. I can only imagine the service rendered when a pediatrician sees from twenty to forty children. My own feeling has always been that if a pediatrician has "money" as an objective, he should quit medicine and go into business.

Sometime I hope some pediatrician will discuss problem mothers rather than problem children. Much could be written on the subject, but perhaps it would, like Mrs. H.'s letter, be largely letting off steam.

ARVID WALLGREN

THE October, 1954, Supplement to the *Acta Paediatrica* is a Festschrift "In Honor of Arvid Wallgren on His Sixty-fifth Birthday, October 5, 1954." It is a volume of 635 pages, containing fifty-two articles by associates and friends of Professor Wallgren in seventeen countries. Dr. Henry F. Helmholz has written the dedication, and a bibliography of the publications of Dr. Wallgren lists 303 items starting in 1915. One group of articles in the volume reflects his scientific interests and work, and another the work of his associates in Stockholm. The remainder, two-thirds of the volume, consists of contributions by invitation, and covers practically every field of pediatrics. As a result of his scientific work and his tremen-

dous interest in international pediatries, "Wallgren" is a "household word" among pediatricians. The JOURNAL extends its felicitations to Arvid Wallgren on this occasion for all that he has done for pediatries in his own country and throughout the world. The volume is a well-deserved tribute and expresses the esteem in which he is held.

CHILDREN'S BUREAU ENDORSED

THE Task Force on Federal Medical Services of the Hoover Commission on the Organization of the Executive Branch of the Government in its recent report recommended that the Bureau be changed from the Social Security Commission to the Department of Health, Education, and Welfare. Further, that the Bureau be continued intact. With these recommendations the JOURNAL is in agreement. While on occasions we have found ourselves in disagreement with some of the medical ideas and proposals advocated by the Bureau, child health is but one of its concerns, and our criticisms have been due to disagreement with the methods proposed rather than with their purposes. During the more than forty years the Bureau has been in existence it has a record of many accomplishments leading to improvement in the welfare of children in the United States.

The text of the report referring to the Children's Bureau is as follows:

The Children's Bureau is one of the few Federal agencies organized strictly on the basis of clientele. Founded in 1912 in a reform era, the Bureau has operated with dedication and with outstanding success. In 1946, when it was transferred from the Department of Labor to the Federal Security Agency (predecessor of the new Department), it was placed within the Social Security Administration.

We conclude that the Children's Bureau does not logically belong in the Social Security Administration for it has important duties outside the welfare field. On the other hand, the work of the Bureau has been so satisfactory and its focus on the whole child so desirable that its functions should not be split among other constituent agencies of the Department.

We believe that the Bureau should be continued intact, and elevated to a point where the Bureau Chief has access to the Secretary. We also favor giving the Bureau authority and appropriations to make research grants.

The Task Force Recommends:

That the Children's Bureau be removed from the Social Security Administration and placed in an administrative position in the Department of Health, Education, and Welfare that will facilitate the major mission of the Bureau. This mission is to take cognizance of the needs of the whole child in the broad fields of health, education, and welfare, support necessary research in the field, and stimulate the utilization of new knowledge by the various agencies of the Federal Government within and outside the Department of Health, Education, and Welfare and in the States.

Members of the Task Force are: Theodore G. Klumpp, M.D., Chairman; Edwin L. Crosby, M.D., Assistant Chairman; Francis J. Braceland, M.D., Otto W. Brandhorst, D.D.S., Edward D. Churchill, M.D., Michael DeBakey, M.D., Evarts A. Graham, M.D., Alan Gregg, M.D., Paul R. Hawley, M.D., Hugh R. Leavell, M.D., Basil C. MacLean, M.D., Walter B. Martin, M.D., James Roscoe Miller, M.D., Dwight L. Wilbur, M.D., and Milton C. Winternitz, M.D.

THE SALK VACCINE

TODAY, Tuesday, April 12, the forms for the June issue close. At 9:30 A.M. a national radio broadcast announced the findings of the Francis report on the experimental mass use of the Salk polio vaccine last year.

That the report was favorable was quite expected, and far from being a surprise to anyone who has been interested. That the Polio Foundation took a "calculated financial risk" last year in continuing to subsidize the six pharmaceutical firms which manufacture the vaccine in order to have a stock pile available in case the report was favorable is quite correct, and praise is due the Foundation in view of the gamma globulin episode. This action was justified and based largely on the results with Dr. Salk's group in Pittsburgh.

That the announced plans and active preparation many weeks ago to enlarge the facilities to manufacture the vaccine commercially were entirely a "calculated risk" has been a difficult explanation to swallow. We have no criticism, but it was rather a definite indication of a favorable report.

Then for weeks a dramatic ballyhoo has been building up. The setting of the date for making the report public on the tenth anniversary of F.D.R.'s death, the invitation to "400 scientists" to its presentation at Ann Arbor, the planned filming of the meeting so that it could be presented on an expensive closed circuit to physicians throughout the country which took weeks of preparation, the publicity given to the free immunization of children in the first and second grades before the results were made known, the advertising literature distributed by the pharmaceutical houses weeks in advance—all these combined to remove any question as to the Francis report being favorable. The real question was, "How good is it?"

It has been a unique experience in medical history and one in many ways we hope will never be repeated. Public interest is natural and quite understandable, but the unprecedented and unnecessary publicity build-up has created difficult problems for the practitioner. The newspapers have given an unusual coverage to a "hope." One shudders to think of the letdown that would have followed an unfavorable report. Only yesterday, Monday, there were discussions in two weekly magazines of quite different character. One in the *U.S. News and World Report* contained the clearest and best constructed graphs of the polio incidence by age and the amount of vaccine available we have seen. The other, in *Barron's*, discussed the vaccine from the financial aspects. The stocks of the companies manufacturing the vaccine have advanced in recent weeks, one almost 40 per cent. *Barron's* states the indicated production cost to the Polio Foundation was about $1.08 per unit of 3 c.c., and puts the projected wholesale price at $3.00. The proposed price to physicians has been between $4.00 and $5.00 per unit.

According to the radio report, the vaccine is safe and from 80 to 90 per cent effective. It is useless to comment in detail on the report. Just how effective it will be will take several years to determine. In the meantime we look with dread to the period which will ensue between today and the time this comment will appear. There are 47,-

050,000 children under 15 years of age and an estimated 28,000,000 units available by June 30. Already it is suggested the immediate use of 2 c.c. per child and the third cubic centimeter months later.

THE GAMMA GLOBULIN PARADOX

THE description by Bruton,[1] in 1952, of a specific clinical entity, agammaglobulinemia, characterized by an absence of γ globulin in the blood serum of patients with a history of repeated severe bacterial infections, led to the study of patients with such a history in many clinics. Similar findings in other patients have confirmed the entity of the condition, and dramatic improvement has followed the use of protective injections of γ globulin. In a recent report, Prasad and Koza[2] defined the syndrome as consisting of a history of recurrent bacterial infections, absence of acquired antibodies, lack of isohemagglutinins, extremely low to absent γ globulin with total serum proteins within normal range, the failure of long-continued antibiotics to furnish protection against infection, and the response to protective injection of γ globulin.

The search for and study of patients with a history of severe repeated infections to find if there was an absence of γ globulin in the blood serum has turned up a surprising paradoxical situation. In some of these patients the γ globulin content was not low or absent, but a markedly elevated level was found. Janeway and his associates[3] reported five such cases at the meeting of the American Pediatric Society in May, 1954, and, in the discussion that followed, similar observations were reported by workers in Minneapolis, Salt Lake City, and San Francisco children's clinics.

Thus we find a paradoxical situation regarding the globulin level in patients presenting the same clinical picture of repeated severe infections. In the one group there seems to be no question but that the low level or absence of γ globulin makes the condition a specific entity, as the protective use of γ globulin quite changes the clinical picture. The second situation is less clear and more difficult to explain, and at present the best that can be done is to theorize. It is a fascinating subject for study and research, and one that potentially is of important significance from a clinical and therapeutic standpoint.

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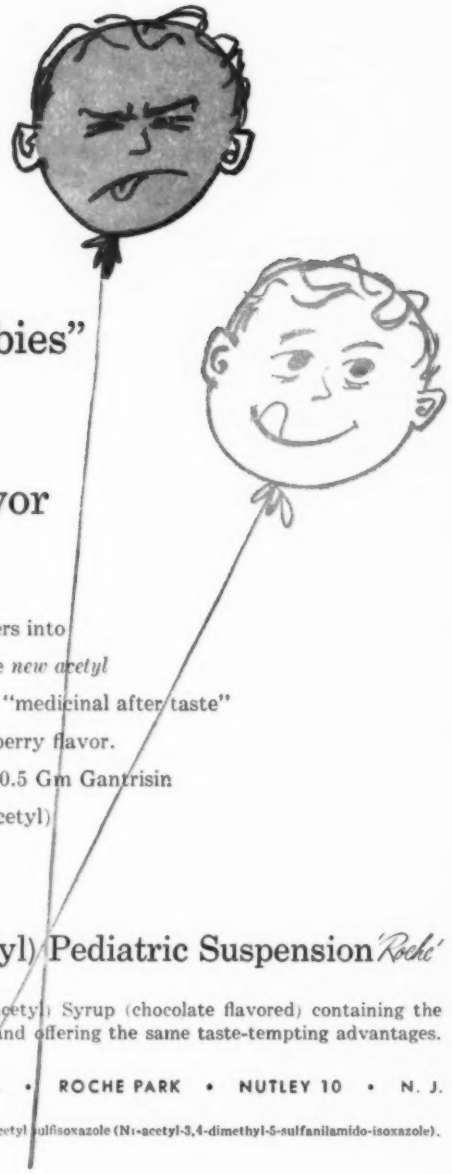

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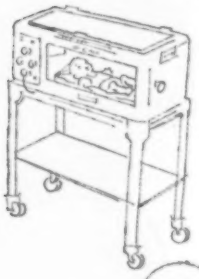

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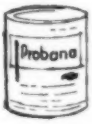

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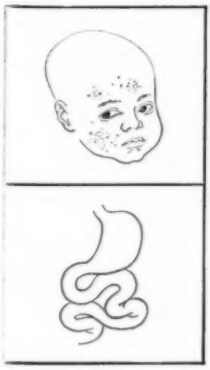

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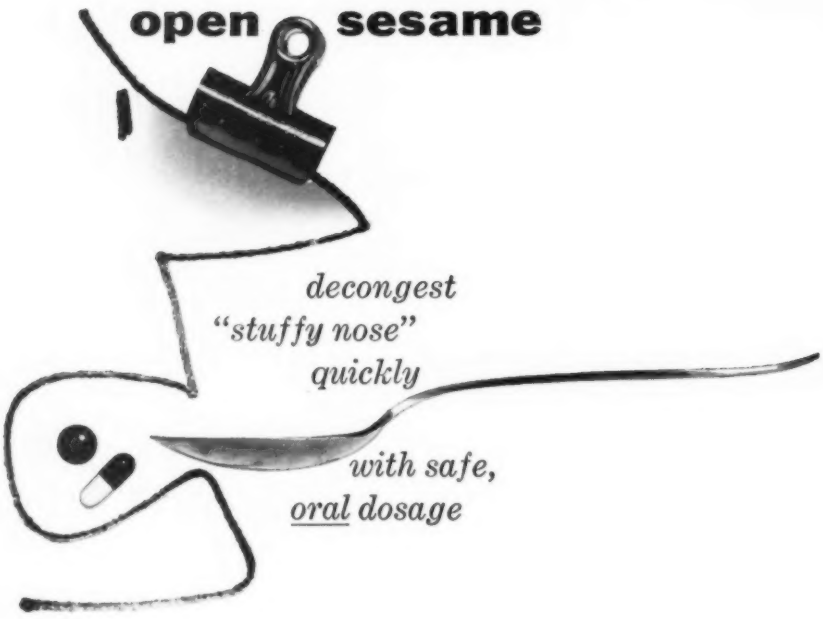

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In feeding tests,* conducted at a widely known institution for child care, 365 infants and children from 10 days to 2½ years old were given daily offerings of Welch's Grape Juice. Infants up to one year were offered 2 to 4 ounces of one-third grape juice to two-thirds water; infants over one year were offered 4 to 6 ounces of one-half diluted grape juice.

Grape Juice was acceptable as a daily routine for periods ranging from one week to three months to 97.6% of the 365 infants tested. None of the infants showed any allergic manifestations.

The doctors who directed the test pointed out in their report that Welch's Grape Juice was not forced but simply *offered.* They consider its long-term acceptance noteworthy compared with other juices. They found it especially useful in cases of allergic manifestations to other juices; where the problem of maintaining correct fluid balance existed; or in the masking of unpleasant medications.

Welch's Grape Juice is made from the pure juice of ripe Concord grapes and is standardized as to strength and sweetness. We confidently suggest it as a helpful and welcome addition to babies' diet.

*Grape Juice in Infant Feeding by Alfred J. Vignec, M.D., and Samuel Weinberg, M.D., *The Journal of Pediatrics,* March, 1954.

LITERATURE AVAILABLE ON REQUEST

Welch Grape Juice Company, Inc.
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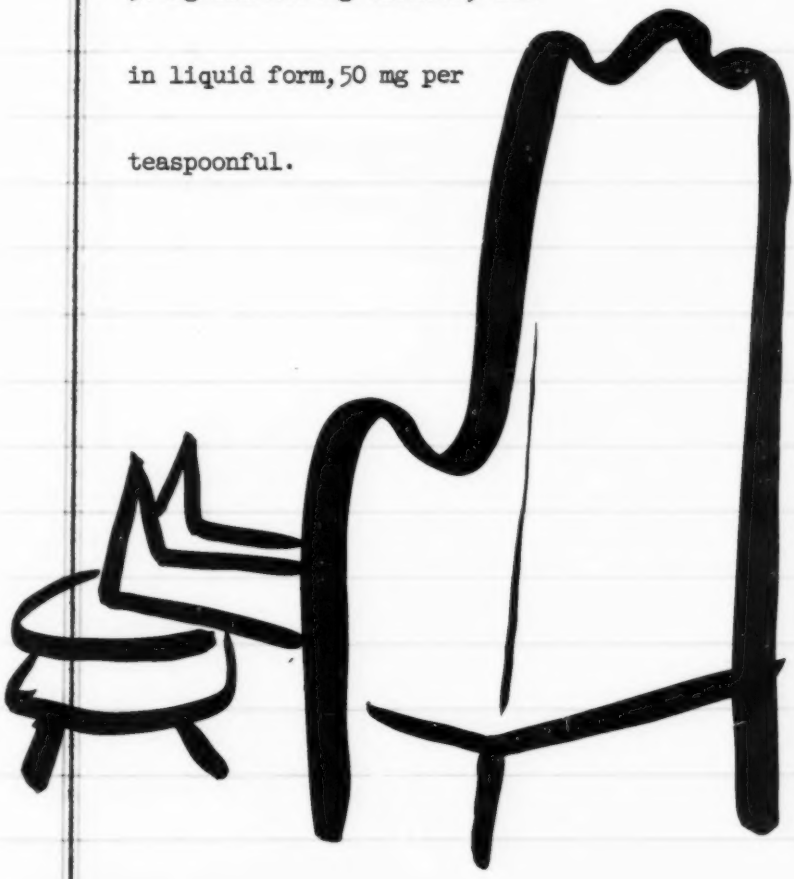
at ease... you may put your own mind at ease as well as calm your patient when you prescribe Noludar as a sedative (or in larger dosage as a hypnotic). There is little danger of habituation or other side effects because Noludar is not a barbiturate. Available in 50-mg and 200-mg tablets, and in liquid form, 50 mg per teaspoonful.



REST COMES BEST to the relaxed

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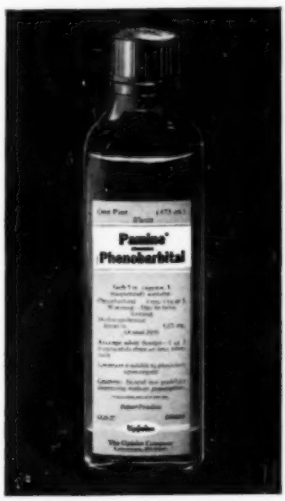

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A	5000 USP Units
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B_1	2 mg.
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B_{12}	5 mcg.
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smooth sedation for the hypersensitive child

MAJOR ADVANTAGES: Induces "natural sleep and sedation without any medullary depression."[1] "One of the safest of all sedatives."[2] Affects no vital function.

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References: 1. West Virginia M. J. 49:292, 1953. 2. Mod. Med. 19:59, 1951.

When you specify the Pfizer antibiotic of your choice *Stress Fortified* with the B-complex, C and K vitamins recommended by the National Research Council, be sure to write SF* on your prescription

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CAPSULES 250 mg.

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ORAL SUSPENSION (fruit flavored)
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The minimum daily dose of each antibiotic *(1 Gm. of Terramycin or Tetracyn, or 600,000 units of penicillin)*
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Niacinamide	100 mg.	Menadione	
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Now Beech-Nut adds a fifth variety to its pre-cooked cereals for babies—*Rice Cereal.*

Beech-Nut Rice Cereal is made from selected rice and rice flour. It is a *one-grain* cereal, especially indicated for infants with a family history of allergy. Bland in flavor and super-smooth, it is readily accepted as a first food and easily digested.

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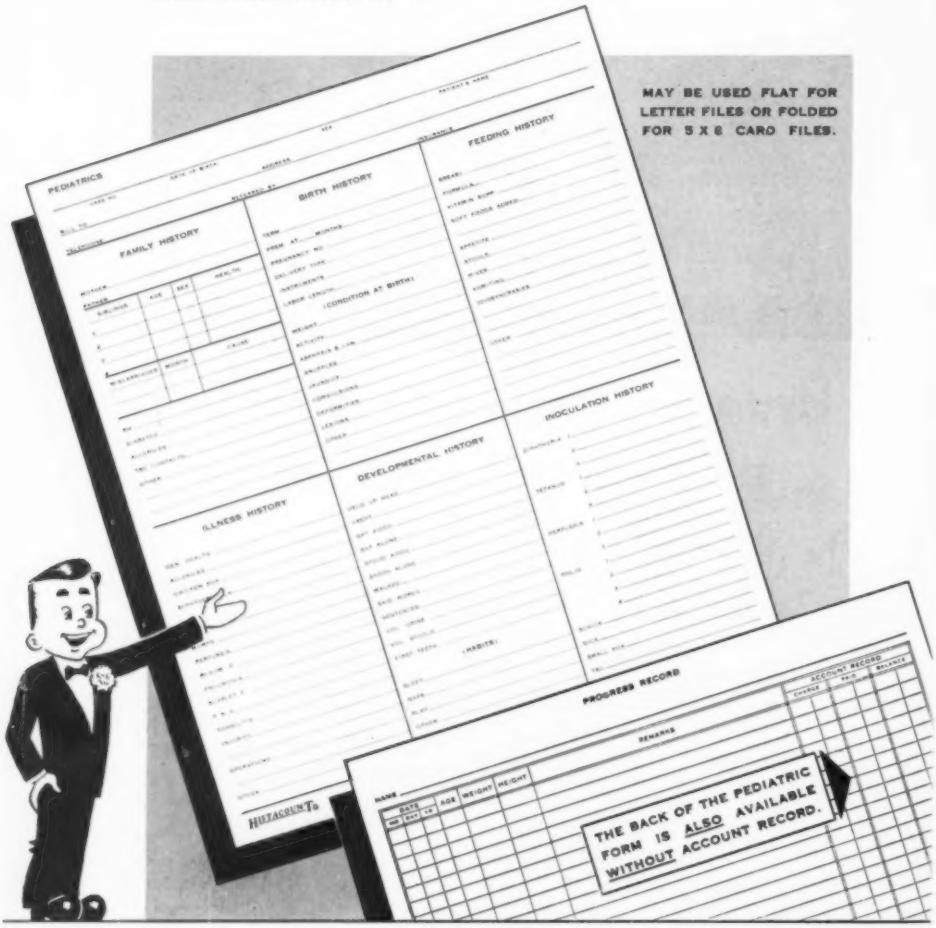

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PINWORM INFESTATIONS

97% CURES[1]

GENTIAN 'E. V.' SUPPRETTES
Rectally

- To break the reinfection cycle
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- To destroy the young and adult oxyurids in the intestinal tract

ALMOST 100% CURES can be obtained in 14 to 28 days in pinworm infestations with combined oral and rectal medication. Using Gentian 'E. V.' Supprettes rectally in conjunction with oral therapy, Allen obtained 97% cures in 77 cases.[1]

1. Allen, Jr., F. P.: A New Approach in the Therapy of Enterobius (Pinworms), J. Pediatrics, 46:155-157 (Feb.) 1955

Actual Size

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In jars of 14 (One complete treatment)

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AQUACHLORAL SUPPRETTE—
Chloral hydrate for rectal use
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For rectal administration
Also w/secobarbital sodium
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Lytren is supplied in 8 oz. cans, with special measure enclosed.

References: (1) Conn, H. F.: Current Therapy, Philadelphia, Saunders, 1954, pp. 338-343. (2) Darrow, D. C.: Mod. Med. 22: 115-116, 1954; personal communications. (3) Harrison, H. E.: Pediat. Clin. North America, 1954, pp. 335-348; personal communications. (4) Cooke, R. E.: Personal communications.

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Phosphorated Carbohydrate Solution

A unique formula for *oral* administration . . . containing no drugs likely to induce untoward effects . . . and stabilized at an optimally adjusted pH, EMETROL has proved dramatically effective in epidemic and other types of functional vomiting.[1-3] In an 18-month study, Bradley and associates obtained excellent responses in 172 children, often with a single dose of 1 to 3 teaspoonfuls. EMETROL is easy and pleasant to take, safe for all age groups.

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---

1. Bradley, J. E., et al.: J. Pediat. 38:41, 1951. 2. Crunden, A. B., Jr., and Davis, W. A.: Am. J. Obst. & Gynec. 65:311, 1953. 3. Tebrock, H. E., and Fisher, M. M.: M. Times 82:271, 1954.

Literature and sample on request

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PORTRAIT OF A POOR APPETITE . . .

When a mealtime masterpiece like this is part of your clinical picture,

prescribe **TROPHITE***

to stimulate appetite and promote growth.

Each tablet or teaspoonful (5 cc.) provides: 25 mcg. B_{12}; 10 mg. B_1

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Thiamine HCl (B_1) ... 2 mg.	Choline ... 20 mg.
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odorless and tasteless

no sting, burn, irritation, or other local reactions

no rebound congestion

especially useful during the allergy season

systemic effects rare in recommended dosage

calibrated dropper for precise dosage

DOSAGE: 1-2 drops in infants under two years, and 2-3 drops in children two to six years.

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- Cherry flavored . . . pleasant and easy to take.
- Xylocaine Viscous has proved valuable in the "dumping" syndrome, hiccup, pyloric spasm caused by peptic ulcer, stomatitis, pharyngitis, esophagitis, acute cardiospasm, pylorospasm in infants, severe vomiting of pregnancy, esophagoscopy, gastroscopy, gastric intubation and gastric lavage.
- Contains 2% Xylocaine Hydrochloride in an aqueous solution adjusted to a suitable consistency with carboxymethylcellulose. Cherry flavored for palatability.

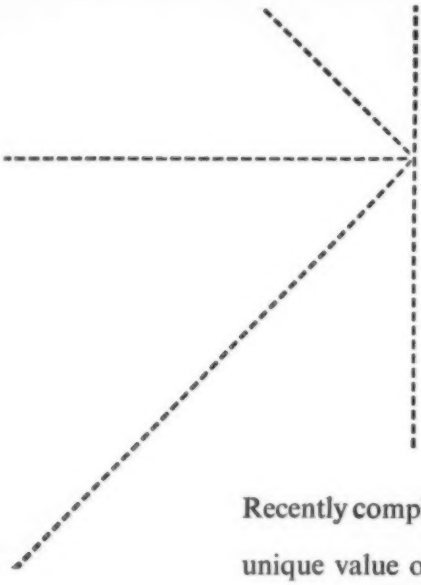
Supplied: In bottles of 100 and 450 cc.
Average Dosage: One tablespoonful, administered orally.
Additional information available upon request

ASTRA

Astra Pharmaceutical Products, Inc., Worcester 6, Mass., U.S.A.

*U.S. Patent No. 2,441,498

Areas of Clinical Study / One of a series



ANEMIA OF INFANCY

Recently completed—1954—studies [1,2] again confirm the unique value of Roncovite (cobalt-iron) in the prevention and treatment of infant anemia.

Clinical results show that routine administration of Roncovite can completely prevent the iron deficiency which so frequently develops in the first six months of life.

RONCOVITE (*Cobalt-Iron*) *has introduced a wholly new concept in anti-anemia therapy. It is based upon the unique hemopoietic stimulation produced only by cobalt. The application of this new concept has led to marked, often dramatic, advances in the successful treatment of many of the anemias.*

SUPPLIED:

RONCOVITE DROPS

Each 0.6 cc. (10 drops) provides:

Cobalt chloride.....40 mg.
(Cobalt 9.9 mg.)

Ferrous sulfate.....75 mg.

DOSAGE:

In children one year or older 0.6 cc. (10 drops); infants less than one year 0.3 cc. (5 drops); once daily diluted with water, milk, fruit or vegetable juice.

EFFECTIVE

"It is a significant fact that none of the... cases receiving iron as well as cobalt required additional iron therapy and that the haemoglobin levels of this group remained consistently and significantly higher than those in any other group after the age of 4 months."[1]

"...there can be no doubt that the average hemoglobin values...are greater in the cobalt-iron (Roncovite) treated group."[2]

PATIENT SATISFACTION

"...the mothers of these anaemic infants frequently stated spontaneously that the children were much improved, with increased appetite and vigour. It seems possible, therefore, that even if anaemia in premature infants does not usually produce marked symptoms, there is a subclinical debility which becomes more evident in retrospect."[1]

SAFETY

"There was no evidence of toxicity in any case under treatment:...There is nothing to suggest that cobalt in any way impairs the general progress or rate of weight gain in premature infants in the dosage employed."[1]

"The babies were closely observed daily for ill effects of the medication while at the premature unit and when they returned for check ups. None of them showed harmful effects despite the large doses...A few of the babies ...have been followed for more than 100 days with no ill effects noted."[2]

1. Coles, B. L., and James, U.: Arch. of Disease in Childhood *29*:85 (1954).
2. Quilligan, J. J., Jr.: Texas State J. Med. *50*:294 (May) 1954.

Bibliography of 192 references available on request.

RONCOVITE

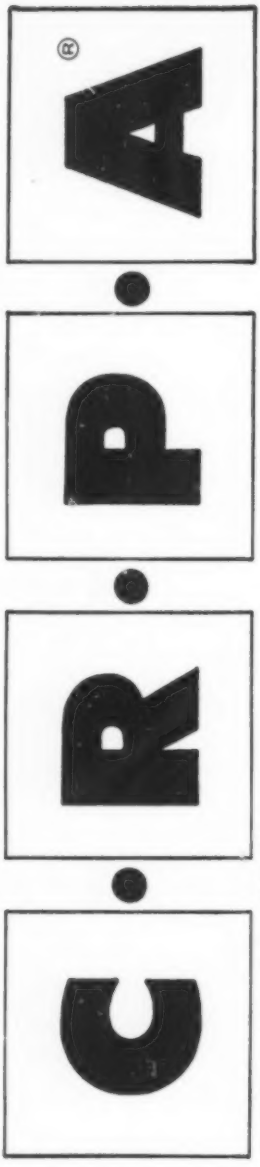
The original, clinically proved cobalt-iron product.

LLOYD

BROTHERS, INC.

Cincinnati, Ohio

In the Service of Medicine Since 1870



(C-reactive protein antiserum, Schieffelin)

PROGRESS ON RHEUMATIC FEVER DETECTION

widely used test for detection of RHEUMATIC FEVER[1]

"The technical procedures involved in carrying out the test are exceedingly simple . . . Except in cases of pure chorea, C-reactive protein is always present in the serum in acute rheumatic fever, and the amount present is more or less proportional to the severity of the illness."

aids considerably in diagnosis of RHEUMATIC FEVER[2]

"Difficulty in establishing the diagnosis usually arises in the milder forms of the disease." Acute phase proteins, such as C-reactive protein, however, "can easily be tested for in the routine laboratory examination . . ."

serves as guide to treatment of RHEUMATIC FEVER[3]

"an extremely sensitive and reliable indicator of rheumatic activity . . . Persistence of CRP in the blood during treatment with antirheumatic agents indicates inadequate suppression of the inflammatory process."

more easily interpreted than sedimentation rate[4]

"an aid in deciding when inflammation has subsided . . . useful in following the course of rheumatic fever in the same sense that the erythrocyte sedimentation test is . . . but is more easily interpreted and has the additional advantage that it is not invalidated by the development of congestion of the liver in heart failure."

as a sensitive test for activity of postcommissurotomy syndromes[5]

"The C-reactive protein was found to be the most sensitive laboratory test for the postcommissurotomy syndrome and the most useful in management . . . The C-reactive protein test is suggested as a useful and sensitive test . . ."

1. Wood, H. F., and McCarty, M.: Am. J. Med. *17*:768 (Dec.) 1954. 2. Adams, F. H.: J.A.M.A. *156*:1319 (Dec. 4) 1954. 3. Stollerman, G. H., et al.: Am. J. Med. *15*:645 (Nov.) 1953. 4. McEwen, C.: M. Clin. North America (Mar.) 1955, to be published. 5. Elster, S. K.; Wood, H. F., and Seely, R. D.: Am. J. Med. *17*:826 (Dec.) 1954.

Supplied: 1 cc vials (30-40 determinations); also Capillary tubes . . . vials of 100; Capillary tube racks.

For complete descriptive brochure on materials and techniques required just send request to:

Schieffelin & Co. New York 3, N. Y.

Pharmaceutical and Research Laboratories since 1794

When you specify the Pfizer antibiotic of your choice *Stress Fortified* with the B-complex, C and K vitamins recommended by the National Research Council, be sure to write SF* on your prescription

antibiotics *Stress Fortified* with vitamins include:

Terramycin®-SF

Brand of oxytetracycline with vitamins

CAPSULES 250 mg.

Tetracyn®-SF

Brand of tetracycline with vitamins

CAPSULES 250 mg.

ORAL SUSPENSION (fruit flavored) 125 mg./5 cc. teaspoonful

Pen-SF*

Brand of penicillin G potassium with vitamins

CAPSULES 200,000 units

The minimum daily dose of each antibiotic *(1 Gm. of Terramycin or Tetracyn, or 600,000 units of penicillin)* **Stress Fortifies the patient** *with the stress vitamin formula* **recommended by the National Research Council:**

Ascorbic acid, U.S.P.	300 mg.	Calcium pantothenate	20 mg.
Thiamine mononitrate	10 mg.	Vitamin B_{12} activity	4 mcg.
Riboflavin	10 mg.	Folic acid	1.5 mg.
Niacinamide	100 mg.	Menadione (vitamin K analog)	2 mg.
Pyridoxine hydrochloride	2 mg.		

**Trademark*

Pfizer

PFIZER LABORATORIES, *Division, Chas. Pfizer & Co., Inc.*, Brooklyn 6, N. Y.

Meat...

and Protein Allowances for Children

Concerned with growth as well as maintenance of tissues, protein needs of children—per pound of body weight and according to age—are one to three times as great as those of adults. These greater needs are set forth in the table of daily protein allowances recommended by the Food and Nutrition Board of the National Research Council,[1] shown below.

Since they deal with the biologic value of proteins and with the vulnerability of children to inadequate protein intake, the following statements accompanying the Board's recent announcement of protein allowances merit attention:

> "The nutritive value of proteins is primarily dependent on the amino acid composition, and the lower biological value of vegetable proteins as compared with animal proteins is well known. The inclusion of a variety of foods, and especially products of animal origin, in the diet assures a satisfactory amino acid distribution as well as sources of other nutrients . . . Recent wide-spread interest in protein deficiency syndromes such as Kwashiorkor disease[2] emphasizes the particular vulnerability of children who receive diets in which the protein content is low and primarily of vegetable origin, even when the total calorie intake is adequate or high."[1]

Meat, when cooked, presents approximately 25 to 30 per cent of high quality protein, easily digested and readily assimilable. Meat is especially acceptable to children and should be a regular item of their diets.[3] It also provides valuable amounts of B vitamins and essential minerals.

Recommended Daily Protein Allowances[1]

Children	*Protein*	Boys	*Protein*
1-3 yrs., 27 lb., 34 in.	40 Gm.	10-12 yrs., 78 lb., 57 in.	70 Gm.
4-6 yrs., 40 lb., 43 in.	50 Gm.	13-15 yrs., 108 lb., 64 in.	85 Gm.
7-9 yrs., 59 lb., 51 in.	60 Gm.	16-20 yrs., 139 lb., 69 in.	100 Gm.
Girls		**Men**	
10-12 yrs., 79 lb., 57 in.	70 Gm.	25-65 yrs., 143 lb., 67 in.	65 Gm.
13-15 yrs., 108 lb., 63 in.	80 Gm.	**Women**	
16-20 yrs., 120 lb., 64 in.	75 Gm.	25-65 yrs., 121 lb., 62 in.	55 Gm.

1. Recommended Dietary Allowances, Report of the Food and Nutrition Board, National Academy of Sciences—National Research Council, Publication 302, Washington, D.C., 1953.
2. Brock, J. F., and Autret, M.: Kwashiorkor in Africa, Bull. World Health Organ. *5*:1 (1952).
3. McLester, J. S., and Darby, W. T.: Nutrition and Diet in Health and Disease, ed. 6, Philadelphia, W. B. Saunders Company, 1952, p. 218.

The Seal of Acceptance denotes that the nutritional statements made in this advertisement are acceptable to the Council on Foods and Nutrition of the American Medical Association.

American Meat Institute

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Upjohn

Infantile colic, regurgitation, food allergy...

Pamine*-Phenobarbital Drops
BROMIDE

Each cc. contains:
Phenobarbital.....20.0 mg. (⅓ gr.)
Methscopolamine bromide.....0.5 mg.
Alc hol.....20%

Dosage (t.i.d. a.c.):
Birth to 3 months..... 5-10 drops
3 to 6 months.....10-20 drops
6 to 12 months.....20-30 drops

Supplied:
15 cc. dropper bottle

*REGISTERED TRADEMARK FOR THE UPJOHN BRAND OF METHSCOPOLAMINE

THE UPJOHN COMPANY, KALAMAZOO, MICHIGAN

AUREO

LEDERLE LABORATORIES DIVISION AMERICAN Cyanamid COMPANY Pearl River, New York

OMYCIN*

HYDROCHLORIDE
Chlortetracycline HCl *Lederle*

Stands on its record!

Seven years of world-wide use . . . more than half a billion doses administered . . . millions of patients restored to normal health, many saved from death—this is the unsurpassed record of AUREOMYCIN.

AUREOMYCIN, the first extensively prescribed broad-spectrum antibiotic, must certainly rank with the major therapeutic agents available.

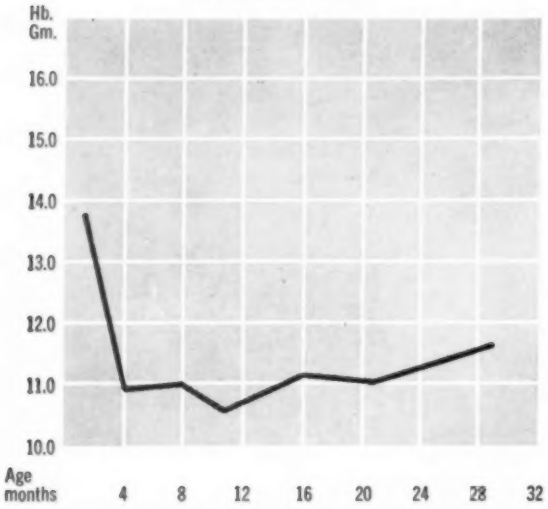
Thousands of published clinical trials have established its efficacy in combating many kinds of infection. Thousands of doctors give it their highest acclaim by regularly employing it in their practices.

A convenient dosage form for every medical requirement. *Lederle*

*TRADE-MARK

"The first additional nutritional need ... of infants is for iron"[1]

IRON DEFICIENCY SHOWN BY HEMOGLOBIN CURVES OF "NORMAL INFANTS"[2]



Means of individual determinations made on a series of "normal" infants and children not given iron supplements.

In the first weeks of life, there is a "relatively rapid transition to an iron-depleted state which is well manifest by 4 months of age and characteristic of the rest of normal infancy."[2]

"Since neither human milk nor cow's milk contains sufficient iron, supplements are necessary by 4 or 5 months of age when the body's stores become depleted."[1]

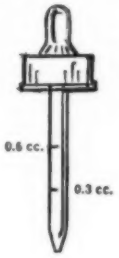
FER-IN-SOL

IRON IN A DROP FOR INFANTS AND CHILDREN

Regular administration of Fer-In-Sol prevents or corrects the iron deficiency so likely to develop in infancy. This pleasant, concentrated solution of ferrous sulfate is well taken and well tolerated by infants and young children.

Only 0.3 cc. of Fer-In-Sol supplies 7.5 mg. of iron—more than the Recommended Daily Allowance for children up to 4 years old. Larger doses provide effective therapy for iron deficiency anemia. Fer-In-Sol® is best given in water or fruit juice between feedings. It leaves no unpleasant aftertaste.

Supplied in 15 cc. and economical 50 cc. bottles with Mead's new, unbreakable plastic 'Safti-Dropper,' calibrated for 0.3 and 0.6 cc.



1 [In addition to the usual breast or formula feedings and vitamin supplements.] Hill, L. F., in Butler, A. M., and Wolman, I. J.: Quart. Rev. Pediat. 9: 63, 1954.

2 Sturgeon, P.: Pediat. 13: 107, 1954.

MEAD JOHNSON & COMPANY • EVANSVILLE, INDIANA, U. S. A. MEAD

high antibacterial and antifungal potency

STEROSAN®

CREAM AND OINTMENT

(brand of chlorquinaldol)

in skin infections due to fungi and gram-positive organisms

A new iodine-free oxyquinoline derivative, STEROSAN has shown favorable results in controlled comparison with other recognized anti-infective medications.*

Of value in virtually all infections due to fungi and gram-positive cocci, STEROSAN is especially indicated in

Dermatophytosis Folliculitis Furunculosis Impetigo contagiosa Impetiginized eczema Infected dermatitides Infected seborrhea Pyoderma Sycosis

The bacteriostatic and fungistatic action of STEROSAN is not hampered by heavy bacterial concentration, pus or organic debris. Sensitization to STEROSAN has not been observed, and primary irritation has been seen only in rare instances.

STEROSAN® (brand of chlorquinaldol) Cream and Ointment, tubes of 30 Gm.

***Tronstein, A. J.: J. Invest. Dermat. *13*:119, 1949.**

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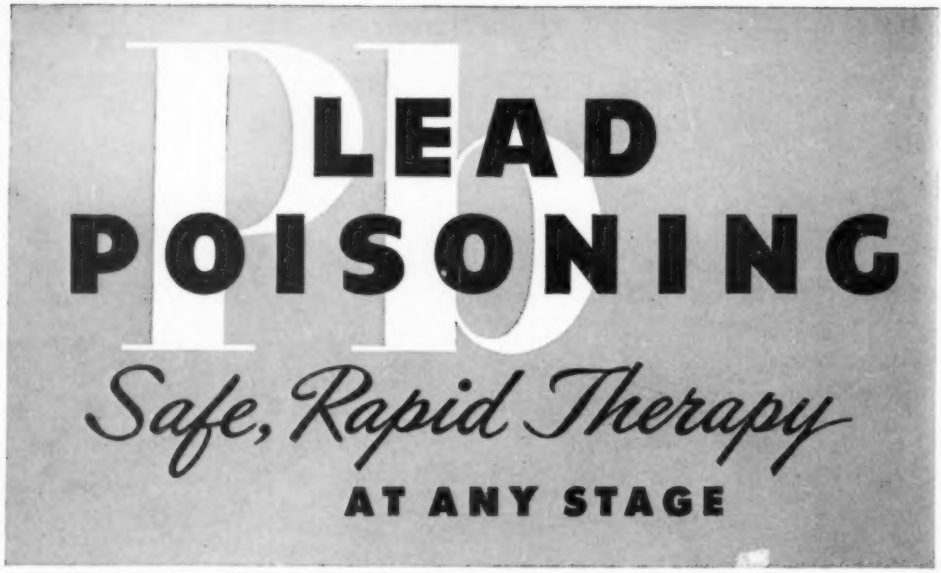
Division of Geigy Chemical Corporation
220 Church Street, New York 13, N. Y.

S6088



Calcium Disodium VERSENATE*

TRADITIONAL methods of treating lead and other heavy metal intoxications are fraught with certain dangers. When heavy metals, particularly lead, are mobilized in the body by methods such as low calcium diet, administration of acids and salts, BAL and similar modes of treatment, damage to the brain, peripheral nerves, and blood-forming organs sometimes occurs from the resulting plumbemia.

In Calcium Disodium VERSENATE, the physician for the first time has a drug which is dependably effective in removing heavy metals from the body *without danger of organ damage*. Heavy metals combine with it to form a nondissociated, water-soluble, intact complex molecule, in which the metal exists in an un-ionized state. This complex is nontoxic to adults and children alike, and is excreted intact.[1]

Patients with acute lead poisoning can now safely be rendered asymptomatic in as little as 5 days.[2,3,4]

1. Rubin, M.: Biological Action of Versene, from Symposium on Complexing Agents, presented at the University of Chicago, Feb. 22, 1951.
2. Sidbury, J. B., Jr.; Bynum, J. C., and Fetz, L. L.: Effect of Chelating Agent on Urinary Lead Excretion. Comparison of Oral and Intravenous Administration, Proc. Soc. Exper. Biol. & Med. 82:226 (Feb.) 1953.
3. Cotter, L. H.: Treatment of Lead Poisoning by Chelation, J.A.M.A. 155:906 (July 3) 1954.
4. Bradley, J. E., and Powell, A. M., Jr.: Oral Calcium EDTA in Lead Intoxication of Children, J. Pediat. 45:297 (Sept.) 1954.

INTRAVENOUS

Indicated to reduce blood and depot lead in acute or chronic lead poisoning, lead encephalopathy, and as prophylaxis against symptomatic exacerbations in chronic lead poisoning.

Complete instructions for dilution and administration for adults and children accompany the ampuls. Available in boxes of six 5 cc. ampuls, each containing 1 gm.

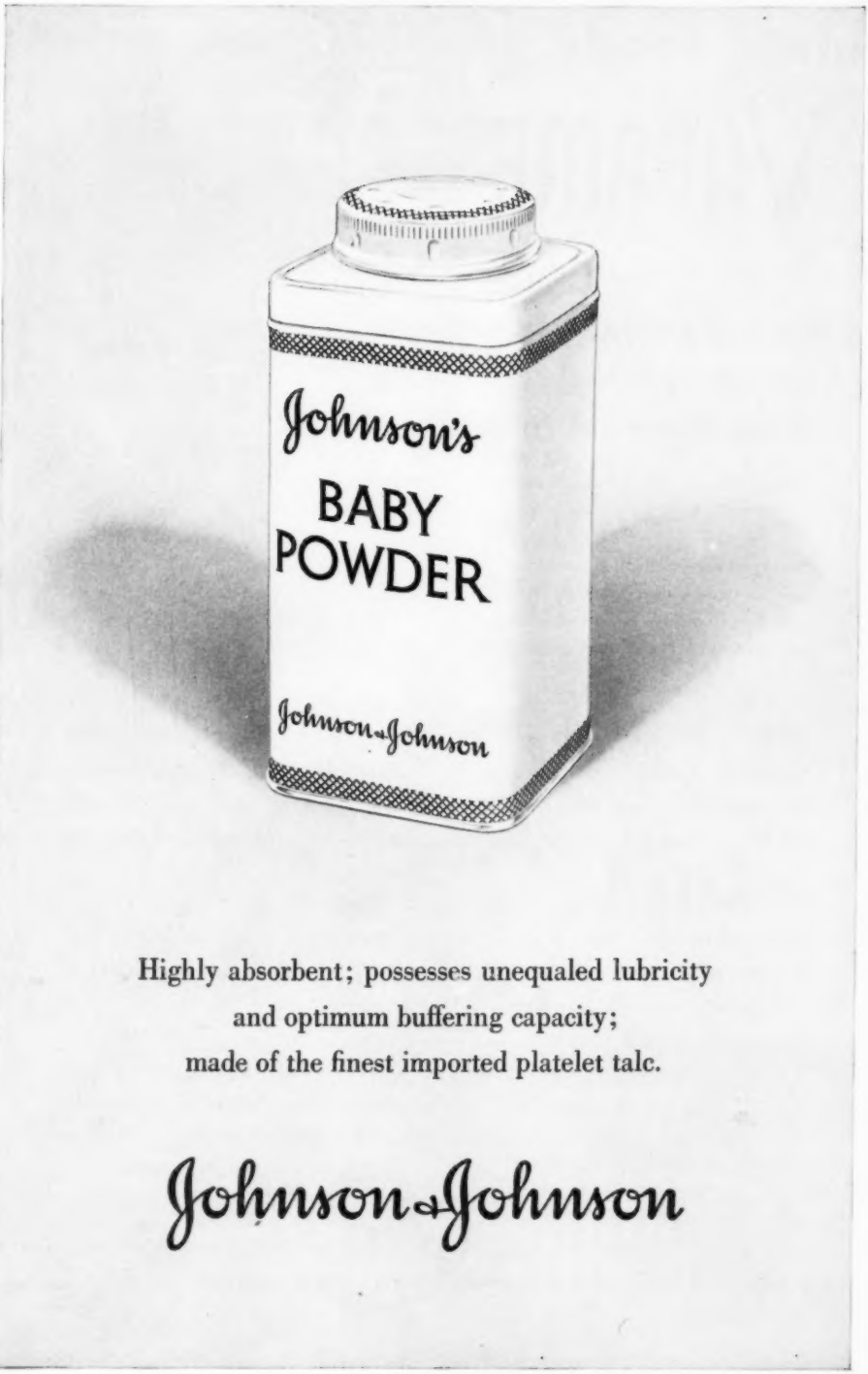
ORAL

May be used as a follow-up to parenteral therapy, to prevent exacerbations, or as sole therapy in lead poisoning not requiring the immediate response afforded by the intravenous medication.

For children, the dose is adjusted on the basis of 2 tablets (1 gm.) per 35 pounds of body weight daily, in divided doses. If tablets cannot be swallowed, they can be crushed and dissolved in milk. Supplied in bottles of 250 tablets, each containing 500 mg.

Riker LABORATORIES, INC., LOS ANGELES, CALIF.

*Trademark of Versenes, Inc.



Highly absorbent; possesses unequaled lubricity
and optimum buffering capacity;
made of the finest imported platelet talc.

Johnson & Johnson

infant foods are *not* "all alike"

VARAMEL

is the only infant formula made exclusively from Grade A Milk*

Designed especially for pediatric use, Varamel is unique in its field and should not be confused with ordinary evaporated milks.

First—Varamel is made from Grade A Milk*, thus assuring a high quality, clean milk from the source of supply to the infant.

Second—Varamel is fortified with sufficient pure synthetic vitamins to provide the daily recommended allowance of all known essential vitamins.

Third—The butter fat is replaced by vegetable and animal fats which are particularly well-tolerated and utilized by the infant.

Fourth—Iron is added to supply 7.5 milligrams per quart of formula.

Fifth—No sugar has been added, so that Varamel is equally as flexible as either whole or ordinary evaporated milk.

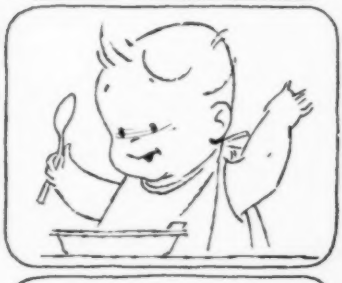
*Made from Grade A Milk (U. S. Public Health Service Milk Code)

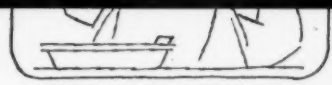
VARAMEL

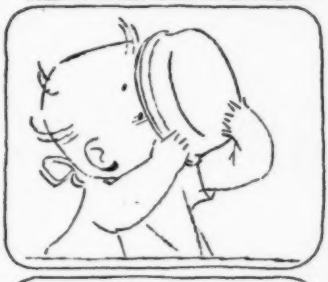
THE BAKER LABORATORIES, INC., Cleveland 3, Ohio
Milk Products Exclusively for Infant Feeding
Plant: East Troy, Wisconsin

VIFORT®
drops

for maximum polyvitamin absorption...







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VIFORT is an aqueous polyvitamin containing synthetic vitamins A and D in small particle size plus five B vitamins; vitamin C; and vitamin E. No fishy taste or odor.

15-, 30-, 60-cc. dropper bottles. Also supplied as capsules.

Samples? **Just write to**

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SPECIFIC FOR "CRADLE CAP"

Softens and separates the scales from the scalp.

Restores normal physiologic function of the scalp epithelium and glands.

CRADOL
WITH DIAPARENE CHLORIDE
DIRECTIONS: To guard against "cradle cap" (crusts and scales on scalp), and scalp infection, gently massage CRADOL into infant's scalp 3 times a week. To relieve "cradle cap": gently massage CRADOL into scalp 3 times daily. At the end of 3 days, shampoo with mild soap and use a fine comb or scale brush. Apply CRADOL daily to help prevent recurrence
CONTAINS: Di-isobutyl cresoxy ethoxy ethyl dimethyl benzyl ammonium chloride in a suitable emulsified base.
FOUR FLUID OUNCES
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another first by the makers of Diaparene® Chloride.

Cradol contains natural oils which protect the scalp against drying elements, lubricates the scalp and prevents recurrence of cradle cap.

Cradol prevents secondary infection. It contains Diaparene Chloride a well recognized and clinically proven antibacterial agent.

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A Subsidiary of Homemakers' Products Corporation

a new anticholinergic solution for G-I upsets in infants and children . . .

A potent anticholinergic agent successfully used in the management of infantile colic, enteralgia, pylorospasm, vomiting and spastic constipation. Also effective in the control of enuresis.

Antrenyl Pediatric Drops are compatible with whole milk, evaporated milk, condensed milk, and orange, pineapple, grapefruit, grape and apple juices. Average dosage 2 to 15 drops a day, according to age of patient.

Supplied: Antrenyl bromide Pediatric Drops, containing 1 mg. Antrenyl bromide per drop; bottles of 5 ml. with dropper.

Antrenyl® bromide
(oxyphenonium bromide CIBA)

Antrenyl®

Pediatric Drops

CIBA
Summit, N.J.

'Homicebrin'

(HOMOGENIZED MULTIPLE VITAMINS, LILLY)

a distinguished member of the Lilly family of vitamins

Little folks often equate growing up with long pants and high-heeled shoes. Actually, of course, healthy growth and development depend largely on balanced nutrition, including adequate vitamin intake. Choice among liquid vitamins is potent 'Homicebrin,' which provides six essential vitamins (including B_{12}).

The appetizing yellow color and delightful vanilla flavor of 'Homicebrin' please the most fastidious youngsters. Just 1 teaspoonful daily usually suffices.

Available at pharmacies everywhere. In bottles of 60 cc., 120 cc., and 1 pint.

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A good milk to travel on . . . and it's available everywhere

You can help young parents enjoy a more relaxing vacation . . . without the anxiety of *hunting* for baby's milk. Recommend Pet Evaporated Milk . . . it's easy to find in grocery stores anywhere. And whenever and wherever it's obtained, Pet Milk is *always* uniform in composition and quality . . . a milk you can *really* depend on.

Favored Form of Milk
For Infant Formula

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MAJOR ADVANTAGES: Complementary effect of three soluble, well-tolerated sulfonamides. More effective blood levels. Sensitization and renal complications are rare. Palatable fruit-flavor.

Supplied: Each 5 cc. (1 tsp.) contains 100 *mg.* sulfamerazine and *200 mg.* each sulfadiazine and sulfamethazine.

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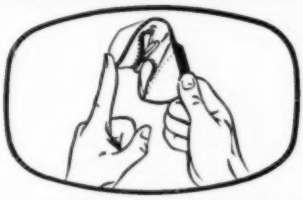

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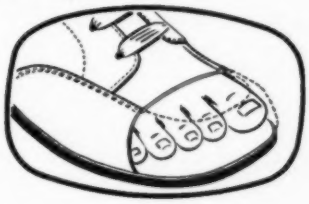

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*Shwachman, H., Children's Hospital, Boston, The Child in Health and Disease, Grulee and Eley, 2nd Edit.

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